Library
of the
University of Toronto

P78-0727.(8)

Digitized by the Internet Archive
in 2018 with funding from
University of Toronto

https://archive.org/details/medjournalNov1937uoft

THE UNIVERSITY OF TORONTO
MEDICAL JOURNAL
(An Undergraduate Publication)

This Journal is published six times a year, November to April, by the Medical Society of the University of Toronto, to serve as a medium for student expression and to bring to the student recent articles of scientific interest.

$1.50 per volume. 25c. per single copy.

ADVISORY STAFF.

Prof. E. A. Linell, M.B., Ch.B., M.D. Manc.
J. Harold Couch, M.A., M.B., F.R.C.S. Edin., F.R.C.S. (C.).
H. H. Hyland, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.),
D. Y. Solandt. M.A., M.D.
Prof. G. F. Marrian, D.Sc., Lond.

EDITOR-IN-CHIEF
W. G. Bigelow, B.A., '38.

ASSOCIATE EDITOR — J. H. Belton, '39.
FEATURE EDITOR — R. A. Mustard, B.A., '38.

ASSISTANT EDITORS
J. C. McCulloch, '39. K. C. Greer, B.A., '40.

WOMEN'S EDITOR — Miss L. McClelland, '38.
MANAGING EDITOR — V. R. Perry, '39.

CIRCULATION STAFF
Manager—B. W. Vale, '38.
Assistants—
W. J. Reid, '38. A. J. Denne, 41.
W. O. H. Perry, '39. D. W. Parker, '42.
E. M. J. FitzHenry, '40.

Address all communications to Room 107, Anatomy Building.

Vol. XV Toronto, November, 1937 No. 1

Editorial

Once again the Journal appears in a fall cover of new design. This, we assure you, is by no means an attempt to change the style each year but a sincere endeavour to acquire a cover which is both attractive and not readily soiled.

The advisory and undergraduate staff of the Journal are working to maintain the quality of this year's Volume XV up to the high standard of journalism found in previous years, preserving it as an article of value to the student. The true value of the Journal is not only derived from what you are able to learn from it but also from what you learn by using its pages to exercise your skill at literary expression.

Forty students contributed articles last year which speaks well for the use that is being made of the Journal by the undergraduates. It is hoped that even more will come forward this year with papers for publication.

* * *

Plans for 1937 - 1938

It is only by attempts to improve the Journal each year that it will live. This year, departments which existed before are being reorganized and new ones installed. The reorganization of the "Abstracts" and "Book Review" column is important. There will be an attempt made to review all articles in the current literature of the month which are of interest to the student. This department will be in charge of Miss McClelland with a capable group of 'reviewers' to assist her. It is hoped that the students in all years will use this column as a guide for their outside reading.

The feature section has planned some new and interesting side lights to cast on our otherwise scientific publication.

The reader may find a list in each issue of the new books acquired by the Medical Reading room during the preceding month.

Further, a section entitled "Medical Society Notes" will bring from the pen of the secretary, Bill Kerr, a monthly account of the doings of the Society and its subsidiary organizations, while a similar column, featuring

medical athletics, shall be managed by Bruce Charles.

Any suggestions for improvement or constructive criticism of existing conditions in the Journal will be thankfully received by the Staff. We encourage letters of student opinion and would be pleased to publish these.

* * *

The University Press

The Book Department of the 'Press' is making an earnest attempt to co-operate with the students of medicine in regard to the sale of books. Their offer of reduced prices and better service, which is by now known to you, is not a scheme to make money—nor is it a scheme to take advantage of their situation on the campus to run competitors out of business but is a sincere desire on the part of its new manager to help and to win the good will of the student.

In this way they have lightened to some extent the heavy financial load which students are forced to carry and for that reason deserve our thanks.

* * *

Professor William Boyd

It is with a great deal of pleasure that we welcome, on behalf of the undergraduates, Professor William Boyd to our Faculty of Medicine. His reputation as a teacher and writer is well known to us all and we feel justly proud that he has chosen Toronto as his new home.

* * *

The Class of '43

The Medical Journal hereby welcomes the members of the first year on their entry into our halls of learning. May your studies prove interesting and may the Journal be of some help to you in the pursuit of these.

Advice is best given by those honoured by that wisest of teachers—Experience. Though we lay claim to very little of that precious attribute, there is one thought we would like to leave with you which should be remembered in the ensuing years of study.

You are going to be undergraduates during a period of time which may see many changes in the practice of medicine. War, political and economic change or State medicine, all of which have shown their heads on the horizon, may bring about sweeping changes in our profession. It is in view of this that we advise you, in your years of study, to prepare as best you can for what the future may hold. Acquire the best and broadest training you can—the better to cope with what new environment you may encounter after graduation.

It would be interesting to be able to peer into the future and witness what changes the practice of medicine is going to experience in this country in the next few years.

If State medicine is going to take its place, what form of administration are we to expect? Many are the plans that are proposed or are being operated now. One has only to look at the number of municipal doctors in the middle west—the organized medical services rendered large industrial and mining centres—or the recent legislative changes in British Columbia. An interesting and successful experiment in community medicine has been carried out in Little Rock, Arkansas, written up recently in

a popular periodical*, which has gone so far as to attract the U.S. Federal Department of Health.

The questions a medical student naturally asks himself are: when is this organized medicine going to become instituted? What form is it going to take? How can one best prepare oneself for the type of work this new method of practice will entail?

This latter question should be borne in mind, but further—a student entering the study of medicine should think past what changes are imminent in the method of rendering medical services and consider which of the branches of medical science are most likely to develop.

Specifically, one immediately thinks of Hygiene and Preventative Medicine as a relatively new field with enormous possibilities. Such a subject as this would become all important in a system of organized medicine. Psychiatry will also strike one as being an interesting science, full of theories with a great future for the practical application of Mental Hygiene arising out of these.

Your study of the basic medical sciences may seem irksome and appear to be too thorough and involve too much detail. Still looking ahead, however, one must realize that it is only with a proper appreciation of the basic principles of physiology, biochemistry and bacteriology that you will be able to fully understand, not only present day clinical medicine, but also new theories of disease and methods of treatment which will appear after you have graduated.

*Readers Digest, August 1937.

In Memoriam

A most promising career was brought to an untimely close on May the eighth, 1937, by the death of James A. Kinnear.

He was born in Toronto on September the fourteenth, 1915, and received his preparatory education at Brown School and the Oakwood Collegiate Institute. In 1934 he began his study of medicine at the University of Toronto, where he was a popular and promising student.

His friends and fellow-students wish to convey their deepest sympathy to his family.

PROFESSOR WILLIAM BOYD.

On the first of September of this year there came from Winnipeg to Toronto one, William Boyd, who had been appointed to fill the Chair of Pathology rendered vacant by the death of his distinguished predecessor, Professor Oskar Klotz. The purpose of the following paragraphs is to give a brief biographical sketch of this recent and very welcome accession to the Faculty of Medicine in our University.

PROFESSOR WM. BOYD, M.D., M.R.C.P. (Ed.), F.R.C.P. (Lond.), F.R.S.C.

Professor of Pathology, University of Toronto.

Professor William Boyd, like certain other eminent personages, including the Archbishop of Canterbury and the Governor General of Canada, is a son of the Scottish Manse. He was born in 1885 in the sequestered town of Portsoy, which is situated on the north-east coast of Scotland. On reaching an appropriate age he was sent to school, first to Trent College near Burton-on-Trent (renowned alike for the products of the school and of the brewery), and then to Torquay on the Devonshire coast, just south of Dartmoor, where during spring and summer the air is filled with the song of the lark, and not far from Babbicombe Bay. Much, no doubt, happened during these earlier years, but little is recorded. Boyd was in the recipient stage—receiving not giving, like a battery that is being charged.

In 1902 he began the study of Medicine in the University of Edinburgh. As an undergraduate he had evidently taken to himself the watchword "excelsior," for he became conspicuous as a climber of mountains, a teller of tales, and at the annual dinner he used to entertain his year with gory and dramatic stories like that of Gunga Din. He distinguished himself in physiology, taking first place in Professor Sir Edward Sharpey-Schaffer's class, and for this he was awarded the medal. No such reward crowned his efforts in pathology, which may be a matter of consolation to the aspiring student.

After graduating he held a number of positions as house physician and wrote his M.D. thesis on the cerebro-spinal fluid. For this he was awarded a gold medal. Bent on improving his knowledge of asylum work he moved north to Inverness. While there he employed his time so well that he was awarded the Gaskell prize and goldmedal in psychiatry and he fitted himself for his next appointment, which was that of pathologist to the General Hospital at Walverhampton.

Then came the war, and 1914 saw the future Professor in France during those early, critical days when the battle of the Marne was being fought and the first battle of Ypres. The impressions then made on him he has divulged

in his first literary effort—"With a Field Ambulance at Ypres." This story, which was widely acclaimed in Canada and the United States of America, is told in the true, if early, Boydian style —graphic, picturesque, dignified, with restrained emotion, and with something of beauty as a background.

In 1917, that is 22 years from now, he was appointed to the Chair of Pathology in the University of Manitoba —its first fulltime professor of that subject. This would seem to be of importance: it indicates that his thoughts were not confined nor his movements hampered by local precedent and tradition. His position was that of a pioneer. Not long after his arrival he was appointed pathologist to the General Hospital, Winnipeg. This unquestionably, was of the very first importance, for added to his teaching position was the source of his future experience and inspiration, both pathological and clinical, and the two appointments marched amicably hand in hand. They were essential to what he has achieved. Doubtless, without either one or the other his energies would have found dynamic expression, but in some direction different from the one taken.

After a year or two in Winnipeg he published his monograph on the cerebro-spinal fluid; a year or two later there followed a Surgical Pathology now in its 3rd edition; later a Pathology of Internal Diseases now in its 2nd edition; and still later a Textbook of Pathology now in its 2nd edition. All of these are widely used wherever English is spoken. The features that characterize this trilogy are their untraditional treatment, their lucid and picturesque expression, their brevity, the fact that they are complete without being exhaustive, the original and beautiful illustrations. In February of this year his most recent book appeared, "An Introduction to Medical Science." It is designed briefly to afford those beginning the study of medicine—and also to nurses—a panoramic view of the subject of their choice.

For the encouragement of any who may feel that they would emulate the subject of this sketch were they but afforded leisure and the exhilarating sunshine of the long Western Canadian winter, it may be remarked that these are not necessary adjuncts—but industry is necessary. His books, in fact, had to be written between the going down of the sun and its rising, because his day was completely filled with teaching and with routine hospital work for eleven months of the year, Sunday mornings included.

Dr. Boyd established in Winnipeg an entirely novel type of teaching museum, the chief features of which are the following: Pathological specimens are mounted in large watch-glasses, set on deep sloping shelves; above each watch-glass is a microphotograph of a stained section of the specimen exhibited in the watch-glass, and below each is the case-history with relevant notes and references. These the student studies while seated and in comfort. And, as you may read in the Journal of the Canadian Medical Association for October — the Medical Faculty Executive Council of the University of Manitoba has declared that this museum shall henceforth be called "The Boyd Museum of Pathology' since Dr. Boyd had made it the unique thing it is."

As might be expected, many distinctions and honours have come the way of Professor Boyd. Universities in Great Britain and Ireland, Africa, the United States of America, and Canada have approached him with a view to his accepting Chairs of Pathology. The honours he, perhaps, prizes most are the fellowship of that select body, the Royal College of Physicians of London, bestowed on him in 1932; the presidency of another select body, the American Association of Pathologists and Bacteriologists, to which he was elected in 1934; and the degree L.L.D. recently conferred upon him by the University of Saskatchewan.

But, Professor Boyd makes opportunity to mingle recreative pleasure

with work, and in this day he has embraced many pastimes, all of them fervently, though not necessarily for long; riding, mountaineering and figure skating have perhaps had their day; swimming, dancing, photography, gardening, golf and badminton are still on the list—and the list is not complete. He has so far eluded fishing and such lethargic sports.

Such is Professor William Boyd.

J. C. B. GRANT, M.D.

THE INTERPRETATION OF ELECTROCARDIOGRAMS*

By F. CARLYLE HAMILTON, B.A., M.B.

Eighty years ago, it was discovered that with each beat of the frog's heart, a change in electrical potential resulted which could be recorded. Not until 1887 was it possible to make such records of the human heart. At that time Waller, using an instrument termed the capillary electrometer, showed variations in the level of a mercury column, caused by the human heart beat. The instrument was not connected with the heart, but to various parts of the surface of the body, which showed a potential difference due to cardiac contraction. While it was possible to record photographically changes in the level of the mercury column, the inertia of the column was so great that only gross changes were discernible.

In 1903, the introduction of an extremely sensitive "string galvanometer" by Einthoven, made practical the recording of human electrocardiograms. In recent years vacuum tube amplification has permitted the successful use of much less sensitive galvanometers. The acceptance of these latter instruments is based upon their ability to duplicate the original tracings of the Einthoven galvanometer. The genius of Einthoven will long be remembered by cardiologists. Many years after the discovery of the galvanometer a friend wrote Einthoven, congratulating him upon the success of his instrument, but stating that while the instrument could write, it was a great pity that it could not also read. Einthoven replied that his galvanometer was not only able to write, but that it read secrets of the heart never before known.

For many years the electrocardiogram has consisted of three recordings, termed the three leads. The first lead is obtained by placing electrodes upon the patient's right arm and left arm. These electrodes are small metal plates which make an electrical connection with the skin through the medium of a salt and water pad, or in some similar manner. The second lead is obtained in the same manner but utilizing the right arm and left leg; and the third lead, from the left arm and left leg. In recent years, a fourth lead has been added. In this lead one electrode is placed over the precordium—usually in the region of the cardiac apex, and the second electrode on the posterior wall of the left chest or upon the left leg. Information is sometimes found in the fourth lead which is lacking in the three conventional leads; more often the fourth lead is merely confirmatory in nature. It is interesting to note that fifty years ago, Waller used all of these leads, as well as several others now considered unnecessary.

When the electrocardiograph is operating, but before the patient in included in the circuit, the galvanometer string casts a shadow upon the moving film which leaves a heavy line termed the base-line, or more technically, the iso-electric line. With the patient attached, a series of deflections above and below the base-line appear, and these recur in an identical manner with

*From the Cardiac Department of the Toronto Western Hospital.

each heart beat. There are five main deflections or waves, and these are arbitrarily designated as the P, Q, R, S, and T waves. To facilitate measuring the height or depth of these waves, the film bears horizontal markings one millimetre apart. Each fifth line is accentuated so that centimetres and half centimetres are clearly seen. The element of time is also recorded on the film, the latter moving at a rate of one inch to the second, or frequently at a faster rate. Vertical lines on the film record time intervals of 1-25 of one second, each fifth line being accentuated so that the time in fifths of a second is more easily recognizable.

An impluse from the sino-auricular node (pace-maker) initiates cardiac contraction. This impulse spreads in all directions through the auricular musculature, causing contraction and a coincident alteration in electrical potential recorded in the electrocardiogram as the P wave. The P wave therefore indicates contraction of the auricles, and it is missing from the tracing if the auricles do not contract as in auricular fibrillation. When the stimulus for contraction is picked up by the auriculoventricular node it passes, after slight delay, onward to the adjoining bundle of His, and thence through the two main branches of the bundle, and their terminal arborizations to quickly reach all parts of the musculature of the ventricles at approximately the same moment, so that a uniform and effective ventricular contraction may result. The passage through the ventricular portion of the bundle and its branches is coincident in the electrocardiogram with the Q, R, and S waves, the latter often being referred to as the QRS complex. Toward the end of this complex the ventricles commence contracting. After a short iso-electric period the T wave appears in the electrocardiogram, and the termination of this wave marks the end of the ventricular contraction. A period of rest ensues, during which the base-line is essentially level, until the next auricular systole occurs. One presenting a normal electrocardiogram. should now reread this paragraph, at the same time examining Figure 1, re- It will be noted that the P, R and T waves are upright while the Q and S deflections (when present) are inverted. Frequently the T wave in the third lead is inverted, without being consider-

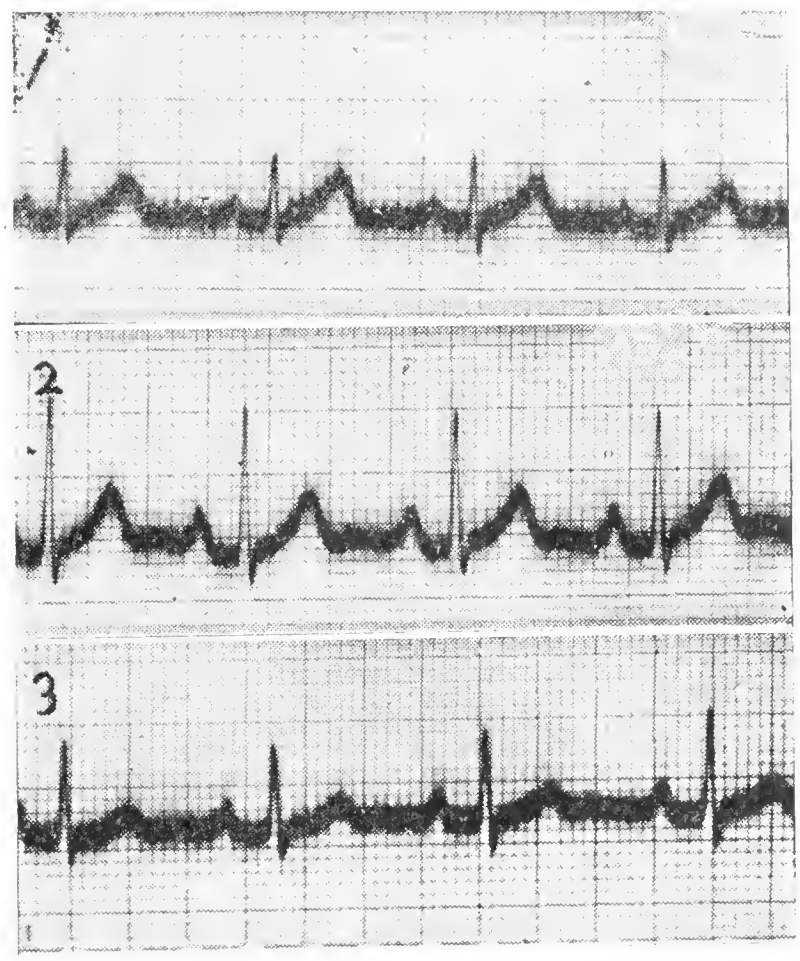


FIGURE 1—The three leads of a normal Electrocardiogram.

ed abnormal. The difficulty of distinguishing the three components of the QRS complex may be avoided, by saying that the main QRS deflection is upward or downward, as the case may be.

All the above mentioned points are significant, and the student should master them before proceeding with the study of abnormal electrocardiograms. In analyzing any electrocardiogram a definite routine should be followed. In this simple manner no abnormality will be overlooked.

The first procedure is to count the rate, for it is of great importance to know if it is within normal limits. The second consideration is to observe the regularity or lack of regularity of rhythm. These two points are mentioned first because they are so often overlooked by the student. After that,

the P waves should be examined, and then the "P-R interval" which is the time from the commencement of the P wave to the commencement of the QRS complex. This should not be over one-fifth of a second. Next the QRS complexes should be examined, in respect to direction, height, and width. The total QRS complex should not last longer than one tenth of a second, and if it is longer in duration than twelve hundredths of one second a very definite abnormality is present. The R-T segment should now be examined to determine whether or not it deviates from the base-line. Finally the size and direction of the T waves should be noted. The time interval by which the T wave follows the QRS complex does not seem of importance. Occasionally after the T wave, a U wave may appear; this is likewise unimportant.

For many years after the introduction of electrocardiography, its chief value lay in the study of cardiac arrhythmias. In recent years it has proved invaluable in the detection of myocardial disease, particularly so in the diagnosis of myocardial infarction. Instead of searching each tracing encountered with view to detecting certain premeditated abnormalities, analysis along the above mentioned lines is much more helpful; and on this account, the description of abnormalities which constitutes the remainder of this article, will follow the above general order.

1. *The Heart Rate*: This should ordinarily be between fifty and ninety. If below fifty, one must think of sinus bradycardia, partial heart block and complete heart block (see below). If above ninety, one must decide between sinus tachycardia, paroxysmal tachycardia (either auricular or ventricular) auricular flutter and auricular fibrillation (see below).

2. *The Heart Rhythm*: The least important arrhythmia in sinus arrhythmia, an irregularity dependent upon the phase of respiration, or in any case being cyclic in type. Very common is arrhythmia due to extrasystoles (premature beats), either of auricular or ventricular origin. Irregularity may be due to partial heart block. In auricular flutter the heart beat is usually regular, but not always so. The most complete type of irregularity is that associated with auricular fibrillation. Other points in the electrocardiographic examination, mentioned below, will decide which type of irregularity is present. The question of heart disease is, of course, not dependent upon the finding of an irregularity.

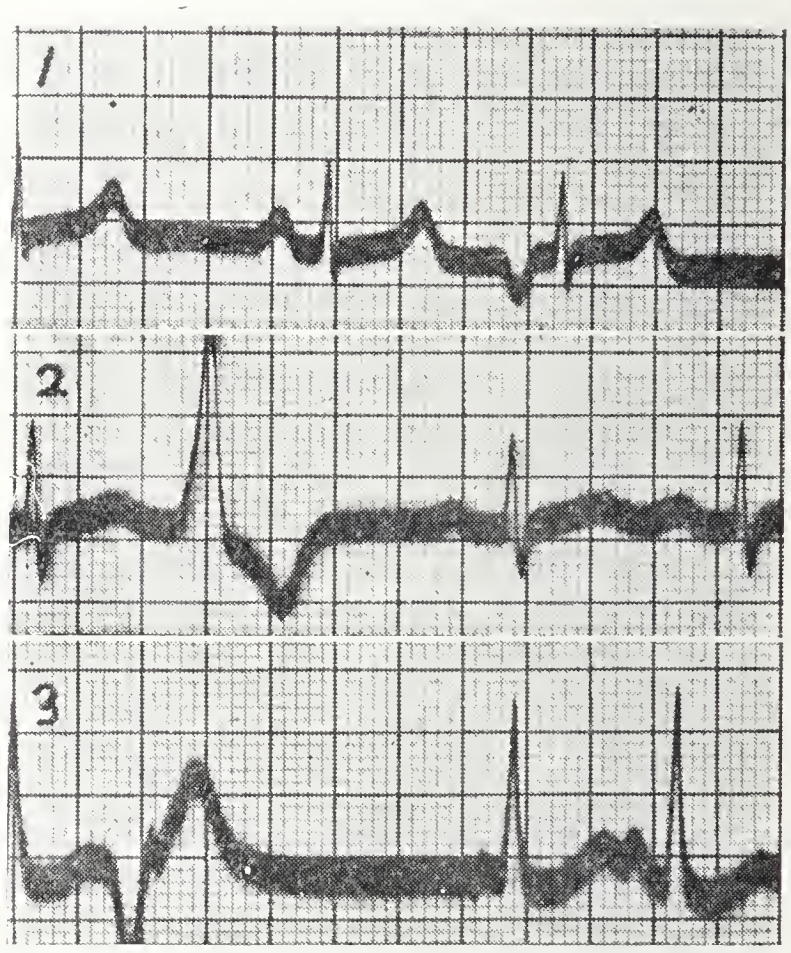


FIGURE 2—Single leads from three cases with extrasystoles. The first shows an auricular premature beat, recorded as an inverted P wave followed by a normal QRS-T complex. The second shows a ventricular Extrasystole—note the compensatory pause which follows. The third tracing shows a ventricular extrasystole, then a normal beat, followed by an auricular extrasystole.

3. *The P Wave*: This is usually upright in all leads. Inversion of the P wave in the third lead is not significant as a rule. If the P wave is inverted in leads one or two, it means that an abnormal pacemaker has supplanted the S-A Node. If an occasional P wave occurs prematurely, it indicates an auricular extrasystole (Figure 2). Such P waves are frequently bizarre in their appearance, but they will be followed

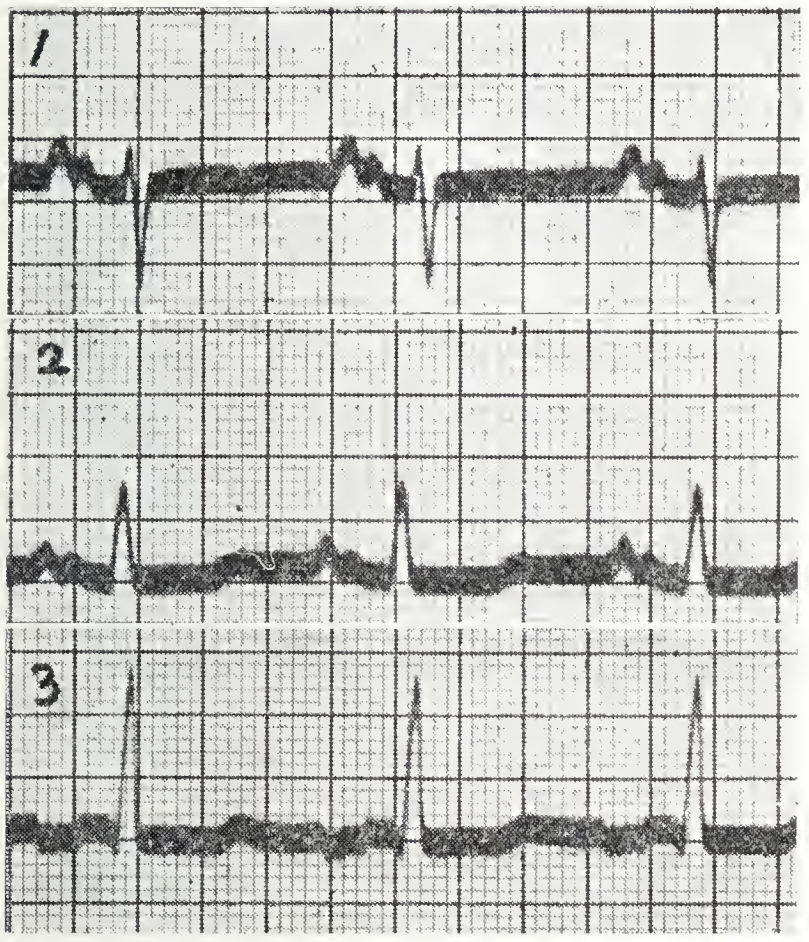


FIGURE 3—The three leads from a patient with Mitral Stenosis. The P waves in Leads I and II are wide and notched. Right axis deviation is present.

(usually) by a normal QRS complex. When a rapid series of successive premature P waves (each followed by a QRS complex) occurs, we have a paroxysm of auricular tachycardia. Such may persist from a few seconds to several days. Practically, auricular extrasystoles and paroxysmal auricular tachycardia are unimportant, unless other evidence of heart disease be demonstrated. Sometimes, in an otherwise normal tracing an entire heart beat will be missing, as shown by an absent P-QRS-T complex; this is termed sino-auricular block and indicates an omission on the part of the pacemaker, but is not usually important. Another point of interest is that in mitral stenosis the P wave in the second lead is frequently large in size and is often notched, (Figure 3). In two important abnormalities, P waves are entirely missing in the electrocardiogram. The first of these is auricular flutter. In this disorder the P waves are replaced by regularly recurring "flutter waves," at the rate of about 300 per minute. Rarely can the ventricle respond to these rapid impulses, so that the ventricular rate is frequently one-half or one-quarter of the auricular rate. Such a tracing is shown in Figure 4; and when once seen, an electrocardiogram of auricular flutter is unlikely to be confused with anything else. The auricular waves in flutter are usually large and of distinctive appearance in the second and third leads, but may be almost invisible in the first lead. The second abnormality in which P waves are entirely absent in the electrocardiogram is auricular fibrillation (Figure 5), a condition much more frequent and important than flutter. Here, the P waves are replaced by rapid small irregular variations in the baseline, which occur about 450 times per minute. Sometimes these small waves are invisible; usually they are seen in some portion of at least one of the leads. The ventricle responds poorly to these rapid irregular impulses, and in a completely irregular manner. The form of the ventricular complexes is not however unusual. The absence of P waves, the occasional appearance in the tracing of varying fibrillary waves,

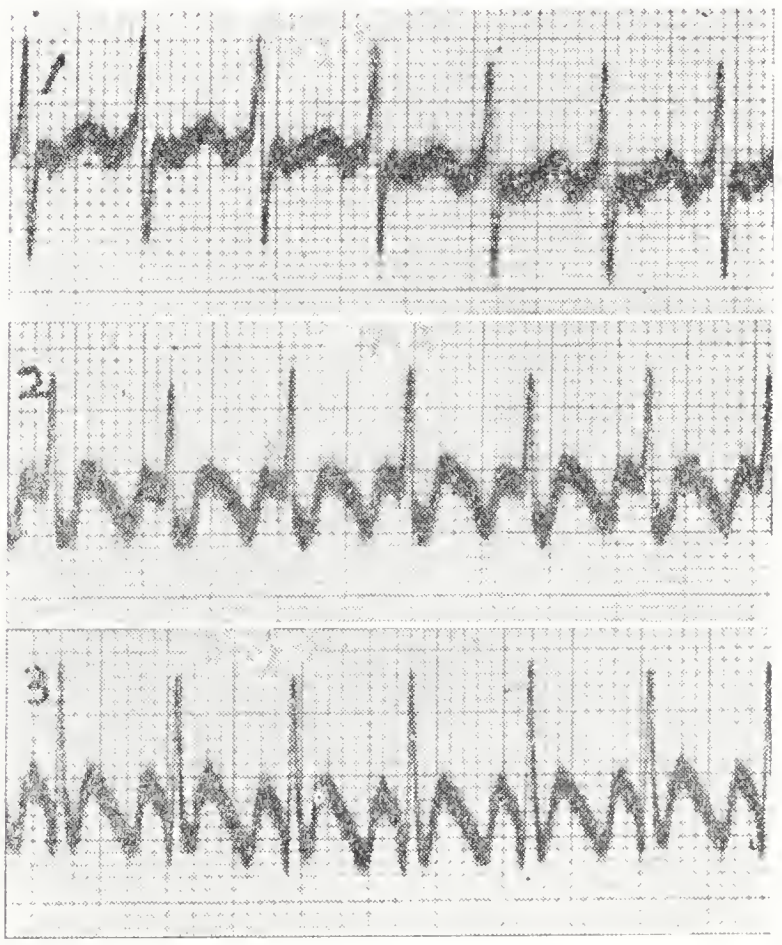


FIGURE 4—The three leads from an electrocardiogram showing auricular flutter, with a 2:1 ventricular response. Auricular rate 340, ventricular rate 170.

and the absolute irregularity of the timing of the ventricular complexes makes the diagnosis of auricular fibrillation certain.

4. *The P-R Interval*: As already stated the P-R interval should not be greater than one-fifth of a second. Where it is prolonged beyond this time, it indicates a delay in the passage of the impulse from auricles to ventricles. This condition of delayed conduction is considered to be the first stage of partial heart block (Figure 6). In it each auricular beat is followed by a ventricular response, but the latter is abnormally, but uniformly late, in appearing. In the second stage of partial heart block, successive P-R intervals tend to increase in length, until a point is reached at which a particular auricular contraction is not followed by a ventricular response at all. A missed beat results, and in the electrocardiogram an isolated P wave is found. The third stage of partial heart block is where such missed beats are a regular occurrence. For instance, for each three

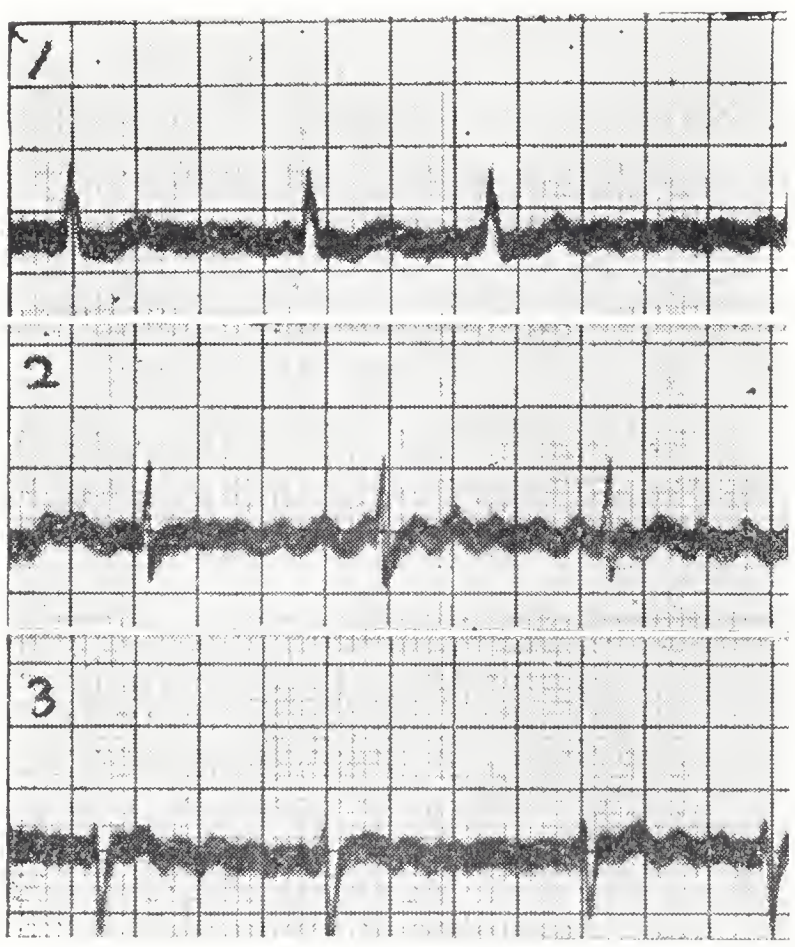


FIGURE 5—The three leads in a case of auricular fibrillation. The ventricular rhythm is completely irregular. P waves are absent, and are replaced by rapid fibrillary waves. Left axis deviation is also present in this electrocardiogram).

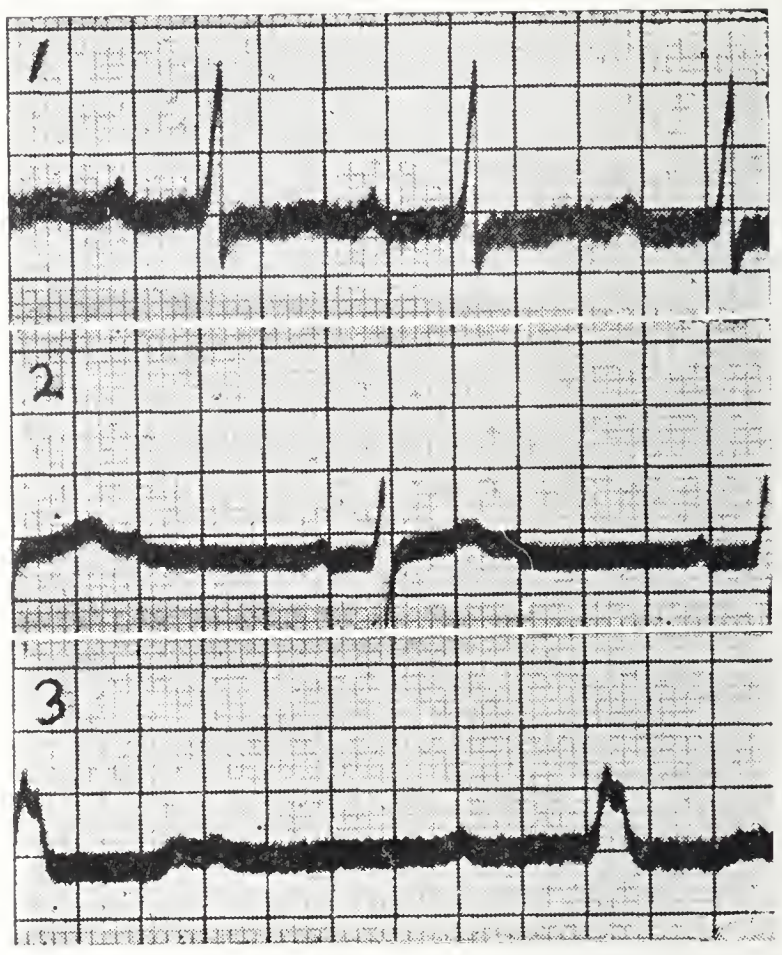


FIGURE 6—Single leads from three cases of heart block. The first shows delayed conduction, the P-R interval measuring about 0.32 seconds. The second shows 2:1 partial heart block—note the P wave immediately after each T wave. The third shows complete heart block and bundle branch block. The auricular rate is 75, and the ventricular rate 33. In searching for complete heart block a much longer strip should be examined than it has been possible to show above.

P waves, only two QRS-T complexes might appear. This would be termed 3:2 partial heart block. Similarly 2:1 partial heart block would be diagnosed if a ventricular response was recorded only after each second auricular beat. Partial heart block may or may not be of serious consequence. On this point some text book such as Lewis' "Clinical Disorders of the Heart Beat," should be consulted. At times complete dissociation between auricles and ventricles occurs. This is termed complete heart block. Fortunately the ventricle takes on a rhythmic beat of its own, usually at a rate of about 30 per minute. Such an electrocardiogram commonly shows a regular succession of P waves at a rate of say 70, with regular QRS-T complexes at a rate of say 30, with no demonstrable

relationship between auricular and ventricular complexes.

5. *The QRS Complex*: In the normal electrocardiogram, the main deflection of the QRS complex is upright in all three leads. In many tracings this is true of the first lead, but in the third lead the main deflection is downwards. This is termed left axis deviation (Figure 5), (formerly called left ventricular preponderance), and is frequently found where the left ventricle is enlarged due to hypertension or aortic valvular disease, and also in normal hearts whose position in the chest approaches the horizontal, as with abdominal obesity and a high position of the diaphragm. Less frequently we find tracings with the main portion of the QRS complex directed downwards in the first lead, and upright in the third lead. This is termed right axis deviation (Figure 3) (formerly, right ventricular preponderance). This occurs where there is relative hypertrophy of the right ventricle, as in mitral stenosis and some types of congenital heart disease, and also in normal hearts, whose position is low and central in individuals with deep chests. In occasional tracings it is found that the maximum deflection of the QRS complex from the base line in any lead, is less than five millimetres. This condition is termed low voltage, and may indicate myocardial damages though not necessarily so. A fairly common and very important QRS disturbance is that known as Bundle Branch Block (Figure 7), inferring block in one of the main branches of the Bundle of His. In this abnormality the QRS complexes are all widened to twelve hundredths of a second or more, and notching and slurring of the complexes are present. In addition, the direction of the QRS complexes in leads one and three is opposite, and the T waves in these leads are also opposite to the main QRS deflections. Such tracings are mainly found in older people with myocardial degeneration resulting from disease of the coronary arteries. Of equal significance are other types of intraventricular block; in all of these tracings the essential feature is a widening of the QRS complexes of the degree above indicated. Mention should next be made of the relatively unimportant ventricular extrasystole (Figure 2). These are easy to recognize in the electrocardiogram, since they consist of premature bizarre ventricular complexes. The individual QRS-T looks much like the complex described under Bundle Branch Block, but it is always premature and not preceded by a P wave. Rarely one sees a tracing in which a series of ventricular extrasystoles occur in rapid succession (about 180 per minute), and this constitutes a paroxysm of ventricular trachycardia. The latter is usually of serious significance, but often responds to prompt treatment by quinidine.

6. *The R-T Segment and T Wave*: These are discussed together because of the significant changes occurring in them after coronary thrombosis. These changes are due to alteration in the heart muscle, and not to the thrombosis

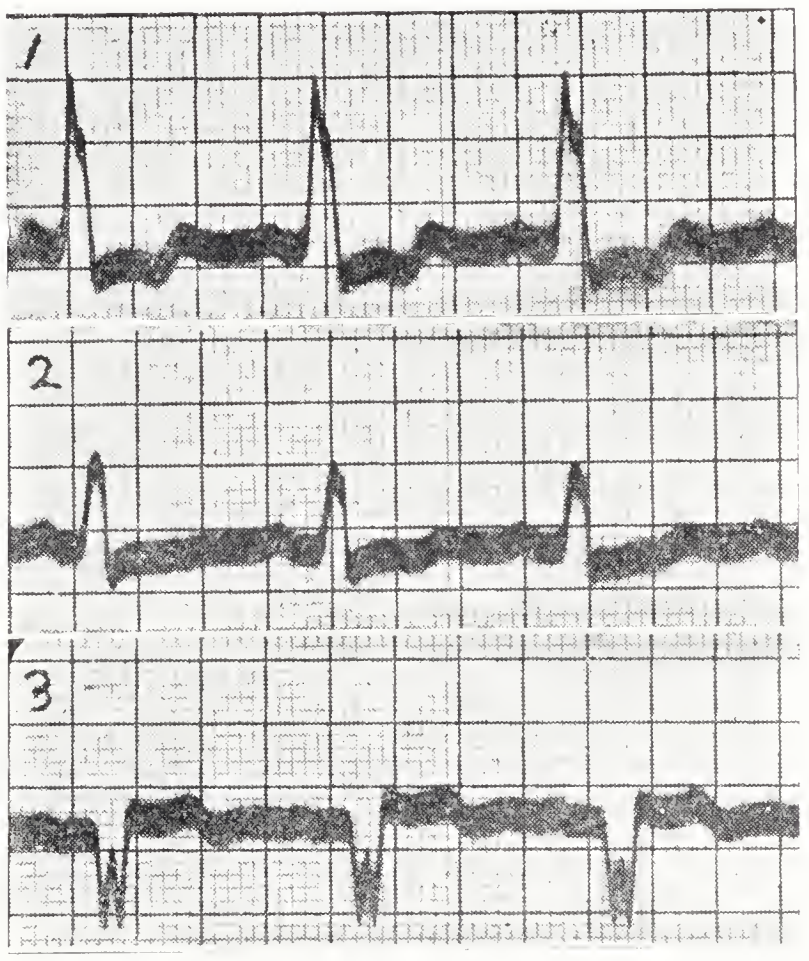


FIGURE 7—Bundle Branch Block. The QRS complexes are widened to about twelve hundredths of a second, and they are notched and slurred. They are in opposite directions in leads 1 and 3, and the T waves are in opposite directions to the main QRS complexes in these leads.

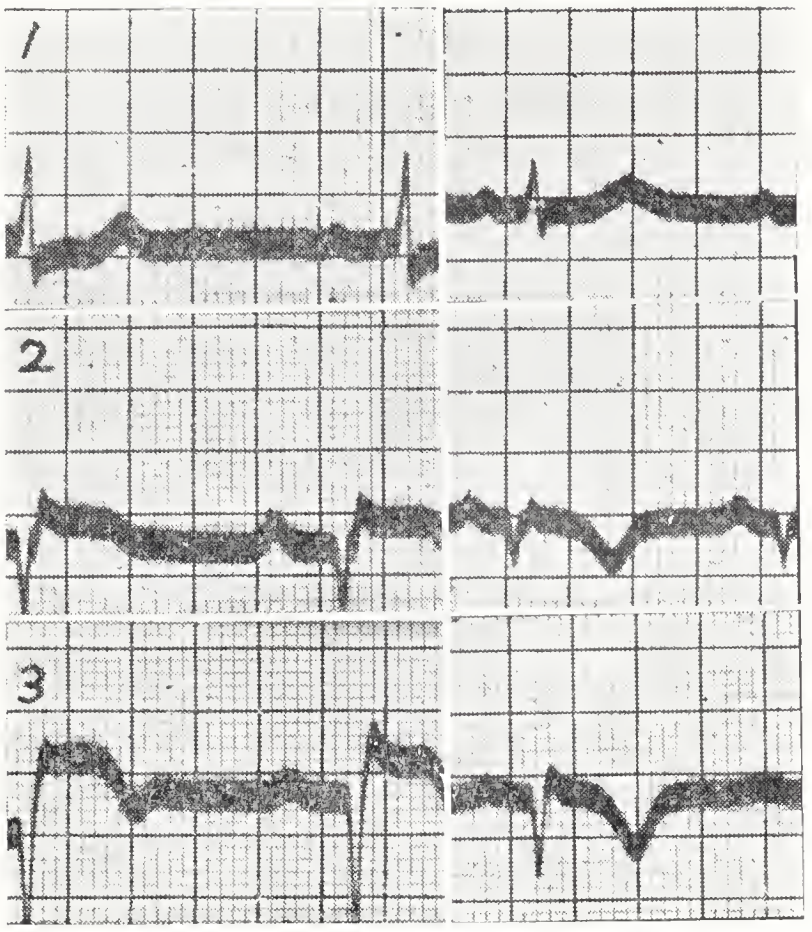


FIGURE 8—Electrocardiograms following coronary thrombosis. Leads 1, 2, and 3, on the left, were taken 24 hours after the attack. Note the low take-off of the RT segment in lead 1, and the high take-off in leads 2 and 3. Leads 1, 2, and 3, on the right are from the same patient, ten days later. The RT segment has returned to the baseline, and marked T waves have appeared in a direction opposite to the original RT segment deviation.

itself. In a typical case, a few hours after the attack, one finds in the first lead either a raising or lowering of the R-T segment (sometimes called the S-T segment) of the base line, and referred to as a high or low take-off from the QRS complex (Figure 8). In the third lead the take-off will be in the opposite direction to that found in the first lead. Usually the T wave is included in this strikingly altered portion of the tracing. In a very few days the raised or lowered RT segment again approaches the base line and a T wave appears, opposite in direction to the original RT takeoff. Over a period of weeks this T wave becomes either sharply inverted, or sharply upright as the case may be. It may persist as such, or the tracing may eventually return to the appearance prior to coronary thrombosis. These changes were first described by Pardee, and are diagnostic of coronary occlusion with myocardial infarction. In many cases of coronary thrombosis, less spectacular changes are found; some times the electrocardiogram shows no significant changes whatever. One should bear in mind that digitalis administration frequently affects the R-T portion of the electrocardiogram, causing a spoon-shaped depression in one or more leads (Figure 9). This must not be confused with changes due to infarction. For many years negative T waves in lead two have been known to be associated with a grave prognosis. The changes in lead two tend to follow those in lead three, and the adverse outlook in these cases is frequently due to the occurrence of a previous coronary thrombosis. Tracings in which the T waves are insignificantly small in all leads are also viewed with suspicion. While serial changes as above mentioned are necessary to the electrocardiographic diagnosis of coronary thrombosis, one may be suspicious that such has prev-

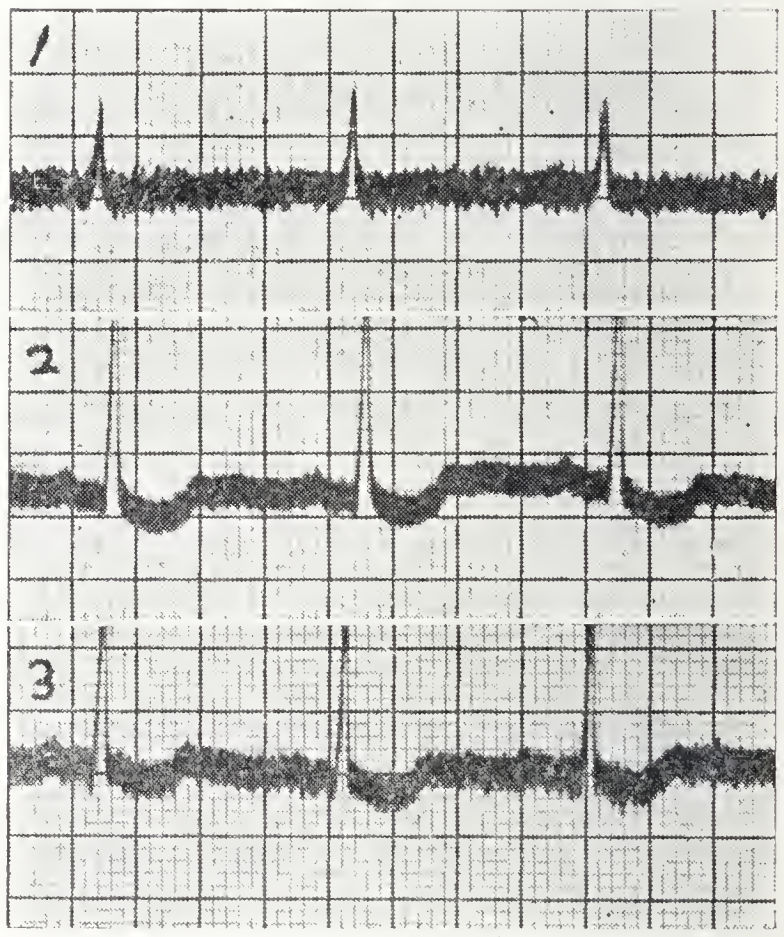


FIGURE 9—Leads 1, 2, and 3, from a patient receiving digitalis. Note the spoon-shaped depression of the RT segments in leads 2 and 3. The T waves have practically disappeared into this depression.

iously occurred in patients whose electrocardiogram shows a very deeply inverted T wave in either the first or third lead.

The fourth lead, mentioned early in this article, frequently shows changes similar to those appearing in lead three, after coronary occlusion. Occasionally diagnostic changes occur in the fourth lead without being evident in the three conventional leads, and in this lies the main reason for its inclusion in the modern electrocardiogram.

Space does not permit the discussion of rarer abnormalities such as nodal rhythm, ventricular escape, etc. However several excellent texts dealing with electrocardiography are readily available for those interested. More than enough has already been said for the average medical student, and my wish is to stimulate interest and thought, rather than to exhaust the details of the subject.

Electrocardiography has a definite place in the study of heart disease. It will never serve to replace a careful clinical examination. In an occasional tracing, a clinical diagnosis is suggested. Such may be the case in tracings following coronary thrombosis. Also, mitral stenosis is most probably present when right axis deviation and auricular fibrillation jointly appear in the electrocardiogram, because of the frequency with which each is met in mitral stenosis. Ordinarily, however, the electrocardiogram must be viewed merely as an adjunct to the cardiac examination. Any opinion based on the electrocardiogram alone is not only unwise but uncertain. While serious electrocardiographic findings are nearly always significant, normal electrocardiograms are not infrequently found in patients with advanced cardiac disease.

Photographs

Our reputation is your guarantee of perfect satisfaction

GEORGE FREELAND
PHOTOGRAPHER

89 Bloor St. West Phone: KI. 0304

THE DEVELOPMENT OF IMMUNITY IN THE HUMAN BODY

By RONALD HARE, M.D. (Lond.)

For the purpose of this short article, immunity may be defined as the mechanism of protection from disease. This immunity may develop naturally or be acquired as a result of infection or artificial immunization. The subject will therefore be discussed under these headings.

I. Natural Immunity.

The great majority of micro-organisms now known cannot cause clinically recognisable infection in man for a variety of reasons, among which may be mentioned their inability to penetrate the skin, to resist the lysozyme of the mucous membranes and to vegetate in his tissues. Thus whole families of organisms such as the Nitrobacteriaceae, so important in economic bacteriology, do not cause human infections; further, many genera, of which the streptococci may be quoted as an example, possess several species which may be highly pathogenic but many more which are quite harmless; and it is highly probable that only a relatively small proportion of the strains encountered in Nature, of species known to be pathogenic, are actually able to cause infection. There is thus a high degree of immunity forming part of the heritage of all human beings, and it is only against the exceptional organism that there is no complete natural immunity.

But human beings are not alike in their ability to resist pathogenic organisms. Thus Jews are unusually resistant to tuberculosis whereas North American Indians and Negroes are unusually susceptible. Environment, too, may play its part in that climate, diet, avitaminosis and fatigue may all render the human host abnormal in his ability to resist a particular organism, while it is well known that such diseases as diabetes and cancer may render human beings unusually susceptible to infection; and there can be little doubt that other factors as yet undiscovered also play their part in depressing or exalting the naturally high degree of immunity possessed by the human race.

II. Immunity Acquired As a Result of Infection.

In its most typical form an infection consists of a preliminary incubation period, a phase of acute invasion, and a period of defervescence or convalescence. It is usually assumed that the immunity of the patient is invariably low during the first phase, mounting during the second, and high during the third; and certainly in the majority of infections, a clinical attack does cause profound alterations in the immunity of the host. These alterations are of three types.

1. The tissues at the site infected undergo alterations which may render them refractory to re-infection without there being necessarily any increase in general immunity. On the other hand, they may apparently return to their original susceptibility. This was demonstrated as long ago as 1895 in the study of haemolytic streptoccocal infections in the rabbit, and is almost certainly applicable to man.

2. Following infection with some micro-organisms such as the vibrio of cholera, the plague bacillus, the viruses of smallpox and measles there is complete immunity which usually lasts for life, whereas with haemolytic streptococci and the viruses of herpes and influenza any general immunity which is produced is usually of relatively short duration. The exact mechanism by which such immunity is produced is quite unknown and that it may be due to the development of antibodies, as enumerated below, is fre-

quently assumed but not invariably or necessarily true.

3. The body of the host reacts by the formation of antibodies to the antigens afforded by the virus or bacterium. Only some of these anti-bodies can be looked upon as being in any sense protective for their possessor, the majority being merely reaction products to the accessory antigens provided by the infecting agent. Thus the virus neutralizing antibodies, the bacteriolysins, and the bacteriotropins are closely associated with virus or bacterial destruction, and the antitoxins with the neutralization of the toxins. But the agglutinins and some of the precipitins almost certainly play no part in the defence of the host. Nor is it possible to state that recovery from an infection depends upon the presence of any or all of these antibodies; in some infections, for example those due to haemolytic streptococci, recovery may take place long before the antibodies one would naturally assume to be essential for recovery have made their appearance; and in other diseases, particularly those of virus origin, infection or even death may occur despite the presence of a strong concentration of such anti-bodies.

But there is now much evidence that immunity to some diseases may be acquired without the host necessarily suffering from a clinically recognisable infection. Thus, during the first months of life the child has a high degree of immunity to such infections as diphtheria and scarlet fever, inherited passively in utero (provided, of course, the mother herself possesses such immunity). This, however, rapidly disappears so that at the end of the first year of life the immunity is at its lowest ebb. Thereafter it tends slowly to rise and by the time adult life is reached a very high proportion of individuals can be shown to be immune to certain infections. Some of these persons will, in the course of the years, have had a clinical attack of the disease in question but a high proportion will not. It is therefore assumed that these latter have had a so-called "subclinical infection" or *infection inapparente*, and have thereby acquired their immunity. Probably for this reason, persons living in areas in which a particular disease is endemic tend to have a higher degree of immunity than others not in contact with the disease. Thus, country bred children are demonstrably less immune to diphtheria than town bred children; and the disastrous effects which occur when a disease such as tuberculosis is introduced into a previously uninfected community are well known.

Nevertheless, this type of immunity only develops against a limited number of infections of which scarlet fever and diphtheria have been most studied; against others such as typhoid fever, gonorrhea and syphilis there is either no such development or it is so irregular as to be, on the whole, valueless.

Lastly, it must not be assumed too readily that the development of immunity by many persons who have not suffered from a clinical attack is due to subclinical infection. The maturation hypothesis of Hirszfeld suggests that it might be part of the development of the individual just as is the development of baldness or the greying of the hair with advancing years.

III. Immunity Acquired As a Result of Artificial Immunisation.

The immunity of animals or human beings can be modified very considerably by two types of procedure. (1) Measures which stimulate the immunity mechanism of the host in such a way as to produce actively an increase in immunity and (2) measures which involve the ingestion of chemical substances or pre-formed antibodies and thus passively increase the immunity.

(1) ACTIVE IMMUNITY.

This is characterised chiefly by the fact that however it is produced it is permanent or semi-permanent. It can be induced by several types of procedure.

(a) *The use of living organisms or*

viruses. In view of the fact that an infection will frequently bring about lifelong immunity and that in childhood, the symptoms may be less serious than in adults, children were at one time deliberately exposed to infectious disease. A similar principle is involved in vaccination against smallpox in which a mild form of the disease induces a high degree of immunity. But, for a variety of reasons, this type of manoeuvre cannot be employed for immunisation against many infections, although Pasteur employed living virus, but of attenuated virulence, for immunisation against rabies, and a similar type of antigen has been recently employed for immunisation against yellow fever. For immunisation against bacterial infections the early workers, such as Haffkine, made use of living organisms; but as a general method the risk was too great and it was dropped for many years until recently when Calmette revived it by using living tubercle bacilli of low virulence for the immunisation of children.

(b) *The use of dead bacteria or viruses.* That a high degree of immunity could be produced by the injection of heat killed organisms was discovered quite early in the history of bacteriology. The degree of immunity produced and the titre of antibodies which made their appearance might be as high or even higher than that produced by an actual attack of the disease. And from animal experimentation we now know that there are very few organisms which, administered in the form of a dead vaccine, cannot bring about sufficient increase in general immunity to protect the host against an otherwise fatal infection with the homologous organism. But we also know that extreme care must be taken to preserve intact the essential antigens in the vaccine. For this reason the strain employed, its virulence and the method employed for rendering it non-infective are all of critical importance. In addition, any immunity produced is only too frequently strictly type specific. Probably for these reasons it is only in a very few instances, such as immunisation against typhoid fever and pneumonia, that immunising procedures of this type are of undoubted value in man, although there is possibility that prophylactic immunisation against other diseases may, in time, be practicable.

Furthermore, the old crude heat killed vaccine is fast losing ground. Antigens extracted from the organisms by chemical treatment are now, experimentally at least, a possibility. And even injection of the material itself which at one time seemed to be an essential part of any immunising procedure may, in the not far distant future, be dispensed with in view of the hopeful results obtained with intranasal insufflation of diphtheria toxoid and the oral administration of typhoid vaccine and the specific carbohydrate extracted from the pneumococcus.

But it is not only in prophylaxis that vaccines have been employed. They have been extensively employed in the treatment of the active disease, and particularly of chronic infections. While their sphere of usefulness is undoubtedly more limited than was at one time supposed, they do produce recognisable changes in immunity and in many instances improvement in clinical condition.

Immunisation against virus infections with killed or inactivated material is also difficult. Nevertheless the Semple method for immunisation against rabies, the Laidlaw Dunkin method for canine distemper, and some current methods of immunisation against yellow fever employ such materials and all are undoubtedly of value.

(c) *The use of toxin freed from organisms by filtration and either used as such or modified by chemical treatment.* The injection of these materials stimulates the formation of antitoxin by the host, frequently in sufficient concentration to prevent infection; but inasmuch as only a comparatively limited number of organisms produce a powerful exotoxin the method is limited in usefulness. Nevertheless in the protection of

whole populations against diphtheria and tetanus it has already proved its value.

(d) *The use of physical agents.* It is highly probable that exposure to ultra-violet light and artificially induced high temperatures modify the immunity apparatus in some as yet unexplained way but sufficient to assist the host in combatting some infections.

(2) PASSIVE IMMUNITY.

This may be induced by two types of reagent, (a) chemical substances and (b) antibodies already present in the blood or serum of another host. This type of immunity is characterised chiefly by its fleeting character, for as soon as the agent employed is eliminated, the host returns to normal.

(a) *Chemical.* It was discovered early in the history of bacteriology that bacteria and viruses could be killed by chemicals and attempts were naturally made to raise the immunity for bacterial diseases by such substances. For a variety of reasons these attempts were unsuccessful, but Ehrlich showed that a *cribrum therapeuticum*, to use his own terminology, was practicable provided the *dosis tolerata* of the drug was greater than the *dosis efficax*. And salvarsan in syphilis, Bayer 205 in trypanosomiasis and sulphanilamide in streptococcal infections are now an important part of our armamentorium.

Of more recent development but in the same category are the attempts to prevent disease by modification of the portal of entry as in the attempted prevention of poliomyelitis by spraying of the nose with chemical solutions.

(b) *Antibodies from another host.* The prevention or treatment of disease by the injection of serum or blood containing antibodies has proved of considerable value in some types of infection. Two types of sera are usually employed.

(i) *Antibacterial or antiviral sera.* Such sera contain antibodies which probably play a considerable part in the actual destruction of the organisms. That is, they contain bacteriolysins, bacteriotropins or virus neutralizing antibodies in such high concentration that even when diluted by the body fluids they can either prevent or cure an infection. The sera come from two sources; from human beings who have had the disease in question and from animals hyperimmunised with the organisms. It is principally in the treatment and prevention of measles that the former are of value and the latter in the treatment of pneumonia.

(ii) *Antitoxic sera.* Sera produced by the injection of toxin and therefore rich in antitoxin, are employed in the prophylaxis and treatment of diseases in which the symptoms are referable to an exotoxin, that is diphtheria, tetanus and gas gangrene. Into the same category must be placed the serum of horses immunised with snake venom and employed for the treatment of snake bite.

The above very bald summary merely sketches the main outlines of what is now a very considerable science and although it may indicate something of what is known it unfortunately gives little or no indication of what is not known. And the latter is still by far the greater part of our inheritance. For we really know very little about immunity and although we can prevent or cure some diseases we really do not know how we do it and there are far more diseases which we still cannot attempt to control. We are indeed, still far removed from Wright's ideal that "in the future of my prediction, the physician will be an immunisator."

THE PATHOGENESIS AND TREATMENT OF TUBERCULOUS EMPYEMA*

By WM. R. SKELLY, M.D.

For many years tuberculous empyema has been the subject of much controversy among thoracic surgeons as to its proper form of treatment and accordingly it is of consideable interest. This, no doubt, has been stimulated by the tremendous strides that have been made in the surgical treatment of pulmonary tuberculosis.

Tuberculous empyema is of much rarer occurence than ordinary post-pneumonic empyema and is secondary to pulmonary tuberculosis in the vast majority of cases. Thus it is more likely to occur between the ages of 15 and 35. In pyogenic empyema following pneumonia the patient has just to overcome the empyema but tuberculous empyema constitutes an extra burden in the tuberculous patient.

Etiology and Pathogenesis

Whether the tubercle bacillus alone can cause an empyema is not clear. Some authors claim that the responsibility may lie with some secondary invader which later dies out leaving the tubercle bacillus in possession. Others disagree with this, drawing attention to the pure tuberculous pus in Pott's Disease of the spine. In a certain proportion of cases of tuberculous empyema, tubercle bacilli can be demonstrated in the fluid either by smear, culture, or guinea-pig inoculation but they are usually in association with other organisms. Of these staphylococcus aureus is by far the most frequent secondary invader. So common is this that the finding of the staphylococcus in an empyema, in itself, suggests that the condition may be primarily tuberculous. The so-called sterile purulent effusions are nearly always associated with a tuberculous process.

Tuberculous empyema can probably only result from the implantation of tubercle bacilli into the pleural cavity from an intrathoracic tuberculous focus. This may occur by direct spread from a subpleural tuberculous lymph gland or from a diseased lung, or by rupture through the pleura of a tuberculous cavity in the lung or a "cold abscess" in the vertebral column. Tuberculous empyema, then, may complicate the following:

1. *Simple tuberculous pleural effusion.* Rich considers that the frequently expressed opinion that tuberculous effusions are allergic manifestations following the lodging of tubercle bacilli in the pleura is erroneous. He believes that in each case a tuberculous focus must have ruptured into the pleural cavity.

2. *Artificial pneumothorax.* While the number of cases in which empyema occurs is small, it is sufficient to be worthy of consideration in the choice of a form of collapse in tuberculosis and should be thought of more especially when only a partial and ineffective pneumothorax has been obtained and it is necessary to use relatively high pressures.

3. *Spontaneous pneumothorax.* This refers to the type that follows the spontaneous rupture into the pleural cavity of a tuberculous cavity near the periphery of the lung. It occurs in from 4 to 10 per cent of institutional cases of tuberculosis.

4. *Pneumolysis*: It is the operation by which pleural adhesions preventing the complete collapse of a lung after artificial pneumothorax, are divided. If done by the closed method there is danger of puncturing the lung with the

*This paper was presented before the Alpha Omega Alpha Society, 1937.

probable development of a tuberculous empyema which may be fetid and contain anaerobes.

The pus of tuberculous empyema has no constant characters. It is usually thin and watery and contains masses of caseous material. Occasionally the fluid is thick and creamy but these cases are probably mixed infections. It is sometimes blood-tinged. Archibald of Montreal divides his cases into three types according to whether the aspirated fluid is sero-purulent, purulent, or very thick as in those with a severe mixed infection. In tuberculous empyema the reparative processes are not so pronounced as in pyogenic types of inflammation. Thus the parietal pleura does not become nearly as thick as that seen at operation on cases of unmixed chronic pyogenic empyema. A broncho-pleural fistula is frequently present and there may be one or more infected sinuses in the chest wall at points where paracentesis has been carried out. If a tuberculous empyema is neglected there may be the formation of empyema necessitatis.

Treatment

In the treatment of tuberculous empyema we are inclined to forget, in our zeal to carry out surgical procedures, that we are dealing with a patient who has pulmonary tuberculosis, either minimal or advanced. Rest, fresh air, good food, and other hygienic measures are of prime importance and should not be neglected.

Thoracic empyema following lobar or bronchopneumonia and empyema complicating pulmonary tuberculosis present two entirely different clinical pictures. In ordinary uncomplicated empyema we can look forward to complete re-expansion of the lung and return to normal function. In tuberculous empyema we are confronted by a more serious problem which presents a vastly different and more dismal clinical picture. Its successful treatment has been one of the most baffling combats with which the thoracic surgeon has had to deal.

In tuberculous empyema each patient must be handled as an individual problem. Before thoracic surgery became one of the great factors in the treatment of pulmonary tuberculosis patients with tuberculus empyema were subjected to costectomy, followed by the insertion of a large drainage tube several inches in length. Purulent drainage continued for months or years and recovery was rare. even in patients with arrested lesions and negative sputum. The type of operation alone prevented sterilization of the pleural cavity, as secondary infection always occurred. If the patient did not succumb to an active spreading tuberculosis he died from a progressive generalized amyloid disease as a result of prolonged chronic sepsis. Surgeons exhausted every means to sterilize the cavity using various dyes, gomenol in oil, and even sterile filtered ox bile. To-day, costectomy and simple drainage would yield no better results unless an effort was made to reduce the toxaemia by frequent aspiration of the empyema cavity and to build up the patient with blood transfusions and iron to a point where an attempt could be made to obliterate the cavity and cure the empyema. It is the general opinion that to cure any empyema, tuberculous or non-tuberculous, we must first of all be rid of the empyema cavity.

Before going into the individual treatment of the various types of tuberculous empyema it might be well to mention the different procedures that are used.

1. **Aspiration.** This may be carried out either alone or by air replacement, and can be supplemented with irrigation. It has been suggested that aspiration be carried out once a week at first. If more than 15 ounces of pus is obtained it is best to replace it with oxygen. Otherwise the patient may become faint or an acute exacerbation of the underlying tuberculosis may occur with consequent stimulation to produce more pus. In the case of mixed tuberculous empyema irrigation of the cavity with antiseptic solutions in the hope of

destroying the secondarily infecting organisms is suggested, but the use of irritating fluids and especially of Dakin's solution, is contraindicated. The latter has little or no action on the tubercle bacillus and may lead to the formation of a bronchial fistula or to haemorrhage because of its corroding action on any exposed diseased area of lung parenchyma. If the pus is too thick to aspirate with ease various modifying agents are used, such as Gauvain's solution (guaiacol, creosote, iodoform, ether, and olive oil), chlorazone or chloramine-T (0.5% solution), or pepsin solution.

2. **Oleothorax.** It is the replacement of pus with an oily solution, usually gomenol in oil. The chief indications for its use are:

(i) Recurrent purulent effusion in the presence of a diseased lung where our aim is to keep it collapsed. It is also indicated in cases where the lung tends to expand in spite of air refills.

(ii) As a preliminary to drainage of a secondary infected empyema.

(iii) The presence of a small bronchial fistula.

(iv) Severe toxaemia, whether from a pure tuberculous or from a mixed infection.

Even when oleothorax fails to cure the patient it often makes a subsequent thoracoplasty safer. It is well to remember that oleothorax is not without danger, occasionally causing a massive pleural effusion or a bronchial fistula, even though it may succeed in closing a small fistula.

3. **Intercostal Drainage** (open or closed). Closed drainage, i.e. drainage which is closed to the outside air, is mostly used to get the patient into better condition for more radical treatment. Frequently an intercostal neuralgia develops due to pressure of the drainage tube on the intercostal nerve lying in the groove along the lower border of the rib above. This often necessitates removing the tube and resorting to thoracotomy and drainage, i.e. resection of part of a rib. As with intercostal drainage it may be open or closed.

4. **Phrenicotomy.** It is the extraction of a piece of the phrenic nerve through an incision at the base of the neck and is used either alone or to supplement thoracoplasty, pneumothorax, or oleothorax. Some surgeons prefer to merely crush the nerve the first time so as to give a temporary paralysis which lasts about 5 months. If it appears to be successful and there are no marked gastric disorders it is then made permanent.

5. **Thoracoplasty** (partial or total). A partial thoracoplasty is the removal of several ribs over the area we wish to collapse. Total thoracoplasty consists in resecting the first eleven ribs from their vertebral attachments to the costo-chondral junctions and is done in two or more stages. It is begun from above and may be supplemented by phrenicotomy. If the cavity is very large and the chest wall unusually thickened, the transverse processes of the vertebrae may be removed with the corresponding ribs. The ribs are stripped of their periosteum and the periosteal beds of the first three or four ribs are often painted with 10 per cent formalin in an effort to delay the formation of new bone. This area is protected by the scapula and the slow regeneration of the ribs allows better collapse of the pleural cavity and lung. The interval between each stage should not be less than three and not more than six weeks according to most authorities. Between the stages and after the operation is completed it is important to remove any effusion that may form or else the cavity will not be obliterated. After operation the chest is tightly bound to maintain compression of the diseased side. Thoracoplasty, in the better hospitals, should not carry a greater mortality than 5 per cent. It is indicated (1) when the lung is diseased, particularly if there is cavitation or much fibrosis, and (2) when the empyema cavity persists in spite of less radical treatment.

6. **Rib Resection and Excision** of granulation tissue and thickened pleura forming the outer wall of the cavity, or the fashioning of a hinged flap from this tissue which is then brought into close contact with the visceral pleura. This is somewhat analagous to "saucerization" when applied to cavities in bone. Dr. Janes recommends that there be wide exposure with packing. This should be removed daily and the surface of the wound exposed to quartz light and, if possible, to sunlight, The smaller furrows will granulate in and close without further interference; the large ones may be closed subsequently with pedicle skin grafts.

From even a brief consideration of these methods of treatment it is obvious that the subject is a rather complicated one. Susman has given us a useful classification of tuberculous empyema. It is based on whether or not the patient has demonstrable pulmonary tuberculosis and whether the pus is purely tuberculous or that of a mixed infection. In those cases with minimal tuberculous lesions the object is to get the collapsed lung on the side of the empyema to re-expand and thus obliterate the cavity. In patients with advanced or moderately advanced tuberculosis he attempts to treat the empyema and still prevent the diseased lung from expanding. The latter cases far outnumber the others. Under no circumstances should open drainage be established in a purely tuberculous empyema. If this is done, secondary infection, the greatest possible catastrophe, is certain to occur. Pleural exudates which have shown no organisms on direct smear and have proved negative on culture should never be drained until it has been proved conclusively that they are not tuberculous.

In those with a *pure tuberculous empyema and a minimal pulmonary tuberculosis*, simple hygienic measures may effect a cure. Simple aspiration is only used when the patient is embarrassed by the accumulation of effusion which may be sterile on culture but yields tubercle bacilli on stained smears. It may be supplemented by air replacement in decreasing amounts and the injection of 5 c.c. of 2% methylene blue to sterilize the cavity and act as a pleural lavage. If re-expansion of the lung only progresses to a certain point and then stops, local thoracoplasty should be performed. If it does not expand at all a total thoracoplasty is indicted and the sooner this is done, the better will be the prognosis. If the empyema is long-standing the outer wall may be excised or made into a flap as outlined above, leading eventually to obliteration.

In the cases with a *pure tuberculous empyema and definite pulmonary tuberculosis*, aspiration and air replacement may be sufficient to keep the lung collapsed and also allow the empyema to heal. If the lung tends to expand in spite of the air refills, or the effusion persistently recurs, oleothorax should be tried. If this procedure also fails, thoracoplasty and phrenicotomy should be performed. If thoracoplasty is contraindicated, closed intercostal drainage and pleural lavage may tide the patient over till his general condition improves. The drainage should be in the anterior axillary line, if possible, so as to get well away from the line of incision of a future thoracoplasty. If the patient does not improve sufficiently, nothing can be done but to leave him with a permanent drainage. An exception may be made here to our rule of trying to keep the lung collapsed. If the tuberculous empyema follows an artificial pneumothorax late in the course of treatment, the lung may have recovered sufficiently to allow it to re-expand as in those cases previously described.

There is always the grave danger of a pure tuberculous empyema sooner or later becoming secondarily infected. For this reason, Archibald and others believe that they should be treated radically from the beginning in order to avoid this complication. When secondary infection has occurred, whether from rupture of a subpleural abscess, the formation of a bronchial

fistula, the occurrence of empyema necessitatis, or by way of the aspirating needle, the picture changes from that of an ordinary tuberculous empyema to one more closely resembling an ordinary case of pyogenic empyema. However, certain differences still exist. In many cases, although the patient may have adequate drainage he still looks toxic and continues to be septic with high fever, rapid pulse, rapid weight loss, etc. This is the finding more usually observed in ordinary pyogenic empyema when there is inadequate drainage. In tuberculous empyema the drainage probably does not affect the tuberculous processes of pleura and lungs. In the event of a bronchial fistula occurring in the course of treatment of a tuberculous pleural effusion or tuberculous empyema, secondary infection does not necessarily always occur at once. Thus, if secondary invasion does not follow, immediate thoracoplasty is advisable.

Now let us consider those cases that have *a mixed tuberculous empyema and a minimal pulmonary tuberculosis*. If the patient's condition is good and the infection is mild, aspiration, air replacement, and irrigation may lead to cure. Usually, however, it only brings improvement. In the absence of a bronchial fistula, closed intercostal drainage and irrigation may be tried. Closed drainage has the advantage that the cavity may be reduced in size, thus limiting the extent of the subsequent thoracoplasty which will probably be necessary. Oleothorax might be tested before drainage is undertaken. The results with this are variable but, as previously mentioned, oleothorax may succeed in closing a small bronchial fistula. In the presence of a large bronchial fistula, rib resection and open drainage is the best treatment according to some authorities. Very rarely the fistula closes and the lung re-expands. Most of these patients finally end up with a total thoracoplasty with or without excision of the outer wall of the cavity or its conversion into a flap. Neither cutaneous nor bronchial fistula is a contraindication, and as a matter of fact, both may be cured by the operation Before the thoracoplasty is done the secondary infection must be reduced as far as possible, by aspiration, frequent irrigations, etc.

We can dispose of the cases with a *mixed tuberculous empyema and definite pulmonary tuberculosis* in a few words. They are given the worst prognosis of all four types. The survivors with few or no exceptions will require a total thoracoplasty. The preliminary treatment is the same as for the others but once the secondary infection is under control the thoracoplasty should be done as soon as possible lest the lung should start to expand. If the patient remains too ill for thoracoplasty, permanent drainage and irrigation is all that is left to do.

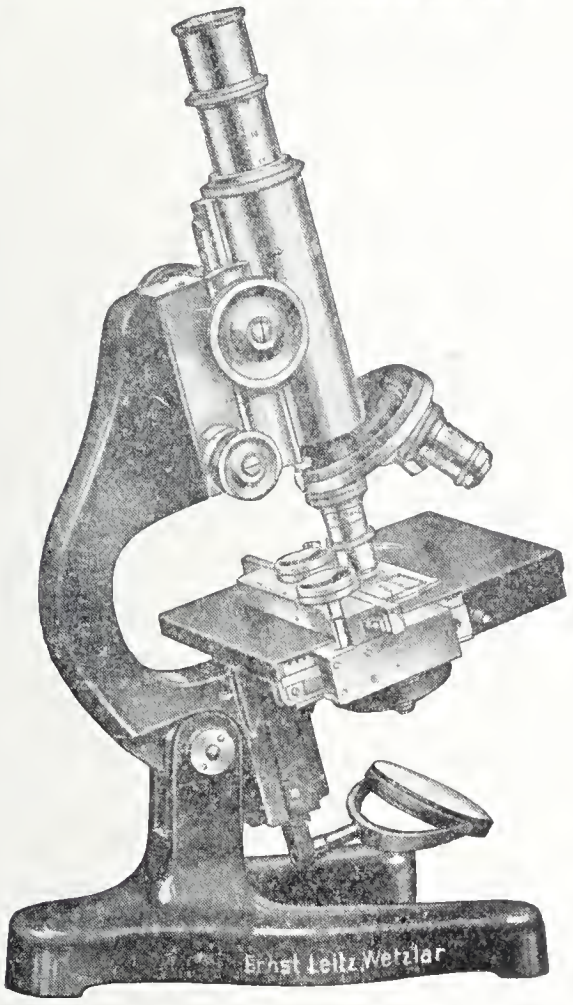
Tuberculous empyema has a mortality between 30 and 50 per cent. In the treatment of simple, uncomplicated tuberculous empyema, thoracoplasty has reduced the disease from one with a mortality of nearly 100% to one with an almost negligible mortality. The chief causes of death in all cases are secondary infection and pulmonary tuberculosis. Less common causes are tuberculous meningitis and generalized tuberculosis. The main operative risks apart from shock, haemorrhage, etc., are aspiration bronchopneumonia and other chest complications.

Summary.

1. Tuberculous empyema is practically always associated with pulmonary tuberculosis and thus the ordinary hygienic therapeutic measures of that disease should be instituted in addition to any surgical procedures.

2. The definite bacteriological or pathological proof of the tuberculous nature of an empyema is difficult.

3. Tuberculous empyema results from the entrance of tubercle bacilli into the pleural cavity, either by direct extension from, or rupture of, some intrathoracic tuberculous lesion.



LEITZ
MEDICAL MICROSCOPES
Recognized the World Over

for their accuracy and quality, embodying the latest and best improvements that optical science can provide.

YOUR SUCCESS IN MEDICINE

will depend not only upon your training and ability, but also upon the quality and precision of your equipment. A microscope will be one of the most important instruments in your equipment both in medical school and in active practice.

WALTER A. CARVETH & CO., 388 Yonge St., TORONTO
CANADIAN DISTRIBUTORS FOR LEITZ MICROSCOPES

4. It may occur as a complication of artificial pneumothorax or pneumolysis. A definite percentage of these become secondarily infected.

5. Many surgical procedures have been recommended for use in treatment and there is much controversy about the relative values.

6. Frequent blood transfusions and iron are by far the best therapeutic agents to improve the general condition of the patient before operation.

7. A sterile empyema should never be treated by drainage until it has been definitely proven to be non-tuberculous.

8. If repeated aspirations and ordinary hygienic treatment fail to obliterate a simple, uncomplicated tuberculous empyema cavity one should resort to thoracoplasty.

9. The treatment of mixed tuberculous empyema by thoracotomy and irrigation is only the preliminary step and should be followed by early thoracoplasty if at all possible.

10. Although the mortality is between 30 and 50 per cent. the prognosis is considerably better than it was before the advent of thoracoplasty.

The author wishes to thank Dr. Janes for his kindness and help in the preparation of this paper.

POTT'S DISEASE — PATHOLOGY AND DIAGNOSIS

By DAVID K. WISE, B.A.

Etiology.

Pott's disease is relatively common in children, about 75% of cases occurring under 10 years of age; it is rare over 35 but no age is exempt. The most common sites affected are the dorsal and dorsilumbar regions of the vertebral column.

The mode of infection is by hematogenous implantation, which implies another active focus elsewhere from which the blood-stream was infected. Usually this focus, primary or otherwise, can be detected and in the majority of cases is found in the lungs. However, at times it is difficult or impossible to demonstrate, because it may be small and so situated as to be symptomless, as may occur in intestinal tuberculosis with mesenteric adenitis. It is also possible that the present spinal lesion was implanted so long ago that the focus of infection has had time to heal and become unrecognizable.

Tubercle bacillemia can actually be demonstrated in some cases by blood culture. In addition, other foci of tuberculous disease of hematogenous origin are found frequently. About half the cases of Pott's disease will display other foci in the spine, other bones, joints or genito-urinary tract.

Morbid Anatomy

The organisms are usually implanted in the cancellous bone of the vertebra near the epiphysis and set up a characteristic reaction resulting in tubercle formation and tuberculous osteitis. This is essentially a destructive process, the bone being slowly infiltrated, absorbed, and replaced by tuberculous granulation tissue. Cartilage is very susceptible to the tubercle bacillus, so that the adjacent intervertebral disks are rapidly destroyed and the disease process spreads across to the adjacent vertebrae. The early lesion therefore consists of destruction of the adjacent surfaces of two vertebral bodies and the intervening intervertebral disks. Destruction of the disk is of importance in distinguishing this condition from secondary carcinoma of the spine, where there is often a strong radiographic resemblance to tuberculosis of the spine but no involvement of the disks. For a long time destruction predominates, but later there may be added an element of new bone-formation, limited in extent.

A periosteal form of the disease is also described in adults, starting usually in the anterior part of the vertebral body behind the anterior longitudinal ligament and spreading around the bones and up and down to neighboring vertebrae. The bone crumbles and the intervertebral disks are destroyed but there is no deep destruction of the vertebral bodies so that angular deformity is not very marked, and the tendency is to produce a gentle curvature of the vertebral column.

Diagnosis

Diagnosis is based on the following points:

1. *Pain*: This is the earliest symptom. Its intensity varies with the duration of the disease, the activity with which it is progressing, and the amount of bone destruction, so that in its earliest stage there will not be a great deal and in some cases there is only discomfort. As the disease progresses, two types of pain are noted according to location, either or both of which may be present:

(i) Local pain: This is situated in the back at the level of the diseased vertibra. Like all pain of spinal origin activity and weight-bearing intensify it and rest relieves it, important points in its differentiation from visceral pain. Percussion over the site of the disease is also painful. Careful questioning may be necessary to obtain the history of

pain, aggravated by standing, walking, stooping, etc., and relieved by lying down in such a manner as to take the weight off the spine. Explosive expiratory acts, like coughing and sneezing, also exaggerate the pain, as do walking downstairs or driving on a bumpy road, movements which suddenly increase the weight borne by the diseased area. These distinctive features must be considered in early diagnosis because the pain does not become disabling until the disease is well advanced. Because of aggravation of pain by movements and weight-bearing the patient learns certain characteristic mannerisms. He walks so as to avoid jarring and on sitting down he carefully eases himself into the chair, his hands supporting his trunk.

(ii) Referred pain: This is experienced in the trunk in segments corresponding in level to the affected area, and is due to irritation of emerging nerve roots. Frequently it may be present without the local pain and if it is one-sided it may be hard to differentiate from the pain of appendicitis or renal stone.

2. *Muscle Spasm*: This is a reflex increase in the tone of the muscles controlling the diseased region, similar to that arising from any other lesion of the musculo-skeletal system. Splinting of the sacro-spinal muscles occurs therefore in the segment affected and for some distance above and below, with reduction of movement in this part, experienced by the patient as stiffness. The patient compensates for the diminished mobility by bending from the hips and not the spine in picking something up from the floor.

3. *Deformity*: The characteristic deformity is a kyphosis which results from destruction and collapse of the vertebral bodies and hence is a comparatively late clinical feature. Owing to the normal lordosis in the lumbar region the deformity occurs much later there than in the dorsal spine and is preceded first by diminution in the lordosis, then complete flattening and finally backward angulation with great collapse of the vertebral bodies. In the dorsal region where the normal curve is backwards the smallest amount of collapse results in a small gibbus and deformity therefore appears much earlier here. However, in either case the condition has been present for a considerable period of time, and the absence of deformity does not necessarily rule out Pott's disease.

The angulation is much less acute where several vertebral bodies are affected, as in the periosteal form of the disease. Lateral deviations of the spine are seen occasionally from unequal spasm or because one side of the vertebral body is more extensively diseased than the other. Secondary changes in the shape of the thorax and abdomen occur as the result of spinal deformity but one does not wait for these in arriving at a diagnosis.

In the cervical region the vertebral bodies are so small that deformity is rarely marked and usually is only noticeable as a shortening of the neck which gives rise to a stunted appearance. Deformity is not seen in the sacral region.

4. *Abscess*: This is a frequent complication of Pott's disease. It usually starts in front of the vertebral bodies where it accumulates until it eventually escapes to one side of the anterior longitudinal ligament or more commonly perforates it. In either case it may track for long distances. These abscesses are like tuberculous abscesses in other parts of the body, not very tender, increasing gradually in size and exciting very little inflammatory reaction so that they are called "cold abscesses." They must not be drained by simple incision because this procedure is likely to result in secondary infection and death from amyloid disease. Inoculation of guinea pigs with pus aspirated from the abscess helps to confirm the diagnosis of tuberculosis. Such an abscess tends to spread along definite anatomical lines, depending on the spinal segment from which it originates:

(i) Cervical: The abscess collects

beneath the deep layer of cervical fascia and bulges into the pharynx as a retropharyngeal abscess. Eventually it may rupture into the mouth or spread laterally to point in the posterior triangle.

(ii) Dorsal: The abscess collects in the mediastinum and does not bulge externally. X-ray reveals a characteristic fusiform opacity surrounding the affected portion. Following the course of an intercostal nerve, the abscess may ultimately point through the muscles of the back or more anteriorly through an intercostal space where it may resemble tuberculous caries of the rib.

An abscess arising from a diseased twelfth dorsal vertebra frequently enters the quadratus lumborum, passes through Petit's triangle, and appears in the loin as a lumbar abscess.

(iii) Lumbar: Usually the abscess enters the psoas sheath, and eventually passes below Poupart's ligament to appear in the femoral triangle where it can be detected by palpation. The great accumulation of pus in the psoas sheath makes complete extension impossible, the psoas muscle going into spasm and producing fixed flexion at the hip joint. In other instances these abscesses may point in the loin or alongside the rectum or through the great sciatic notch.

(iv) Lumbosacral: The abscess lies over the sacrum or more commonly deep in the iliac fossa as an iliac abscess. The pus collects within the sheath of the iliac muscle and sooner or later points above the outer part of Poupart's Ligament in the neighborhood of the anterior superior spine.

5. *Radiographic appearance*: X-ray is a very valuable aid in diagnosis. Good lateral view are desirable and are of greater value than anterior-posterior stereos. In the early stages there are clear or blurred, irregular areas of destruction which involve the adjacent surfaces of two vertebral bodies, while the intervening intervertebral disk is thinned. In adults, the destruction frequently does not advance beyond this stage, but in children it often goes on to complete or almost complete destruction of one or more vertebral bodies with complete absorption of the intervening disk; the bodies are seen to be collapsed, and altered in shape, and angularity is easily demonstrable. With advance of the disease the destructive phase gives way to the healing phase where new bone-formation may succeed in fusing the remnants of the diseased bodies into a single pyramidal mass, revealed as such by X-ray. Abscesses are also diagnosed radiographically since they cast a spherical or spindle-shaped shadow covering several vertebral bodies in the diseased area when seen in an antero-posterior view.

6. *Paralytic symptoms*: These form an uncommon complication, usually a late sign following deformity and rarely occurring outside of upper dorsal Pott's disease. Occasionally it may be an early sign of the condition. It is not as a rule due to bony pressure for although angular curvature may be so marked that the cord is actually compressed, it arises so gradually that it does not lead to paralysis. Paraplegia is usually caused by the protrusion from the collapsing vertebral body of a mass of inflammatory tuberculous material backwards through the posterior longitudinal ligament into the vertebral canal where it lies outside the dura mater and may compress the spinal cord. Less frequently it is due to pressure from the development of tuberculous pachymeningitis or from a sequestrum which has been displaced in the collapse of the body. As the disease subsides the tuberculous abscess is absorbed and the pressure symptoms disappear.

The pressure begins in the front of the cord so that the motor tracts are affected first. The earliest signs therefore are difficulty in walking, frequent stumbling, and incoordinate movements of the lower limbs, going on as the disease progresses to true spastic paraplegia. Sensory disturbances are rare and occur only in severe cases, when they are usually accompanied by incontinence of urine and feces.

INFANTILE SCURVY

By FRANK B. THOMSON, '39

Etiology.

The first record of infantile scorbutus was that made by Glisson in the seventeenth century. Barlow and Cheadle in England were the first to contribute to our knowledge of the pathology underlying the clinical picture, which had itself been previously described by Moeller of Germany. Holst and Frolick in 1907 were able to produce scorbutus in animals by feeding deficient diets. Further effort by many workers has proven that scorbutus is caused by the prolonged use of a diet deficient in vitamin C.

The use of pasteurized milk or proprietary foods without adequate addition of antiscorbutics is the cause of infantile scorbutus. Scorbutus rarely occurs when raw milk is fed or when pasteurized milk plus antiscorbutics are used.

Incidence.

Scurvy rarely occurs in infants less than four months old, the reason being that the new born infant has a considerable store of vitamin C acquired during intra-uterine life. The change of diet at the end of the first year makes the occurrence of vitamin C deficiency unlikely so that scorbutus is uncommon after fifteen months. Six to ten months is the usual period of occurrence.

Rickets is frequently present as an accompanying condition. Both being diseases due to a dietary deficiency, it is not surprising that scorbutus and rickets should occur together.

The susceptibility to scurvy varies markedly in different individuals. There is apparently a constitutional factor present, the nature of which is unknown.

Recently it has been shown that there are, amongst artificially fed infants, many subclinical cases of vitamin C deficiency.

Pathology.

In infantile scorbutus the lesions are confined to the *bones* and *blood vessels*. The essential etiological factor responsible for the pathological changes in scurvy has been shown by Wolbach to be the defective formation and maintenance of the intercellular cement substances of the body.

The lesions in the blood vessels are manifested by haemorrhages in various parts of the body, and are thought to be due to a deficiency in the intercellular material which holds the endothelial cells of the capillaries together.

Subperiosteal haemorrhage is the most common manifestation of the haemorrhagic tendency. These are most frequent near areas of rapid bone growth, the distal ends of the thigh and leg bones being the commonest sites. The upper ends of the thigh and arm, as well as the skull, are also frequently involved. These haemorrhages are often bi-laterally symmetrical. They start near the epiphyseal line and split the periosteum off the diaphysis as they develope. This never spreads to the perichondrium.

Haemorrhages in other places occur with varying frequency. Haemorrhage into the gums is common, into the orbit or viscera it is rare. Haemorrhage into the joint cavities never occurs. Cutaneous ecchymoses or haemorrhage from a mucous membrane are seen at times. Microscopic blood from the urinary tract occasionally occurs but this is rarely so in the G. I. tract. Intracranial haemorrhage is very rare and is the only complication to be dreaded.

The lesions in the bones in scurvy are in brief: (1) alteration in endochondral ossification; (2) change in subperiosteal ossification, and (3) epiphyseal separation.

The normal course of endochondral

ossification is altered in each of three areas. First, there is a decrease in the zone of proliferating cartilage. There are few mitoses and poor order in the columns of cartilage cells. Secondly, the zone of provisional calcification (epiphyseal line) is increased. This is the area in which the cartilage cells have died and the intercellular cartilage has been calcified. This calcified cartilage is normally surrounded by ingrowing osteoblasts and replaced by bone. In scurvy osteogenic activity is so decreased that this does not occur. The result is that the epiphyseal line is broader, less vascular and more irregular than normal. Thirdly, there is a decrease in the osteogenic activity of the osteoblasts on the diaphyseal side of the epiphyseal line which results in the trabeculae in this area being thin and scarce and the bone very weak. The osteoblasts in this area multiply and replace the normal marrow for a considerable distance from the epiphyseal line, with a tissue which looks like ordinary connective tissue. There is some trabecular atrophy throughout all parts of all the bones due to a general decrease in osteogenic activity.

The periosteal changes are mainly two. First, there is an increase in the number of osteoblasts with, as elsewhere, a decrease in their osteogenic activity. Secondly, there are subperiosteal haemorrhages as described above.

Epiphyseal separation to varying degrees is common. The separation is due to a cleavage occurring usually in the wide and brittle area of provisional calcification (epiphyseal line). This temporarily interferes with bone growth, but with proper treatment complete recovery is usual.

Symptoms.

The clinical picture presented in infantile scorbutus is not constant. There are a dozen or so symptoms, none of which are constantly present and any one of which may be present to a varying degree. It is proposed to discuss in turn each of the components of the symptomatology, and finally to indicate their relative frequency of occurrence.

The presence, previous to the outset of local symptoms, of a period of indisposition, fretfulness, pallor and loss of appetite is commonly found. This is usually recalled after questioning.

The first local symptom to attract attention is tenderness of the legs. This usually has an insidious onset and may be accompanied by swelling at the joints.. The knee and ankle joints are the first to be affected.

Fever occurs only in the severer cases and runs up to 102-3° F.

Epiphyseal separation, either complete or partial, is often present when the scorbutus is marked. The lower end of femur or tibia are again the usual sites involved, the upper end of the humerus commonly also. The predisposing cause is the increase in width and the brittle nature of the area of provisional calcification; the immediate cause being a slight trauma resulting in a partial or complete fracture in this area. The diagnosis can be made by X-ray, or by the persistence of pain for two or three days after treatment has started.

Changes in the gums are rare unless the teeth have erupted or are about to erupt. Only those of the upper jaw are affected, and of those the upper incisors in particular. The gum is swollen, vivid, bleeds easily, and in some cases may even ulcerate. Pain may interfere with the taking of food.

Involvement of the costochondral junctions are sometimes found. These may be merely tender or there may be epiphyseal separation resulting in a backward displacement of the sternochondral plate. The sharp, tender ends of the ribs form a scorbutic rosary and are readily palpable.

The blood picture is nearly normal in early scorbutus. In the later stages, however, a secondary anaemia develops and the haemoglobin drops to 35-40%. There is no change in the leukocytes.

Bleeding from mucous membranes

is an occasional symptom. These haemorrhages are small but frequent. They occur more commonly from the roof of the mouth or nasopharynx and are rare from stomach or bowel. If blood or mucus is found in the stool dysentery is the likely reason. Microscopic blood in the urine is found in one-third of cases but gross haematuria is less common.

Petechiae and ecchymoses in the skin are common near the bone lesions. They may be wider spread, however. Miliaria is frequently an associated condition. If any skin trouble occurs during the scorbutic state the lesions are haemorrhagic.

Due to bone lesions in the lower limbs a typical "scurvy posture" is frequently assumed by the suffering infant. This posture involves semi-flexion of the thighs and legs and outward rotation at the hips.

Due to the pain of subperiosteal haemorrhage and epiphyseal separation a condition of pseudoparalysis may occur. The child lies in the scurvy posture and no voluntary movement of the lower limbs takes place.

The constitutional disturbance resulting from the accumulated effect of the various pathological conditions in scorbutus is variable in degree. Many cases present no nutritional disturbance whereas others have marked fretfulness, poor sleep, pallor, anorexia, and loss of weight.

Having discussed the various symptoms of infantile scorbutus it remains to indicate their relative frequency of occurrence. The following list gives one an idea:

Tenderness of legs	84%
Fever	75%
Swelling of legs	71%
Gum changes	66%
Scurvy posture	57%
Skin ecchymoses	33%
Microscopic haematuria	33%
Pseudoparalysis	14%

It is evident, therefore, that the more severe the case under consideration, the greater will be the number of pathological conditions that will manifest themselves clinically, and the more complete will be the symptomatology.

The X-Ray Picture.

By means of the X-ray it is possible to diagnose scorbutus very early, even before the development of haemorrhages. Due to trabecular atrophy the shaft has a ground glass appearance. The epiphyseal line is broad due to the increase in the zone of provisional calcification. This is especially so at the lower ends of the femur and tibia. There is a zone of rarefaction immediately beneath the epiphyseal line due to the marked decrease in osteogenic activity in this area.

Later in the disease, or in severe cases, the above signs become more prominent. In addition, epiphyseal displacement or separation and the presence of spurs of bone attached to the epiphyseal line are seen in the picture. The latter are caused by ossification of the adjacent subperiosteal haemorrhages.

Following treatment the area of rarefaction fills and bone forms under the elevated periosteum. The subperiosteal haemorrhages are gradually absorbed and after a year the X-ray picture becomes normal.

Rickets, occurring as an accompanying condition, complicates the X-ray picture greatly and makes the diagnosis more difficult.

Diagnosis

The diagnosis of infantile scorbutus is not difficult if the following essential pictures are kept in mind: age incidence, extremely tender legs, spongy swollen gums, swelling near large joints, haemorrhagic tendency, and the history of long use of boiled or pasteurized milk or of proprietary foods. There are a few conditions with which it is occasionally confused however. They will be mentioned in turn and a brief statement made as to the chief differences in symptomatology which should

enable a differential diagnosis to be made.

1. Articular rheumatism: does not occur usually under five years of age.

2. Poliomyelitis: confused because of pseudoparalysis in scurvy. In scurvy there is more hyperaesthesia, the reflexes are present and painful stimulation causes movement of the limbs.

3. Acute arthritis or epiphysitis: confused with scurvy because of the fever at times present. These two conditions are rarely bi-laterally symmetrical, have a leucocytosis, the swellings fluctuate, there is pus on aspiration and they do not present the X-ray picture of scorbutus.

4. Syphilitic osteochondritis: occurs before four months.

The diagnosis of scorbutus is most difficult when there are no changes in the extremities. However, a good X-ray will often enable a diagnosis to be made.

If all other measures fail, improvement under antiscorbutic treatment indicates the diagnosis.

Treatment

The occurrence of infantile scorbutus may be prevented by giving antiscorbutic food to all infants getting boiled, pasteurized, dried, or evaporated milk. Orange juice is the best antiscorbutic; this should be given one teaspoonful daily at first and the amount increased to the juice of one orange as quickly as the digestive ability of the child will permit.

The treatment of an existing case is best accomplished by giving four ounces of orange juice per day. If there is much gastro-intestinal disturbance the giving of 100 mg. of ascorbic acid daily by mouth is recommended. If vomiting is severe one can inject the ascorbic acid intravenously. The various gross pathological conditions present require no local treatment.

Course and Prognosis

Death from scurvy is a rare occurrence. Such complications as malnutrition, digestive trouble, or secondary infection may, however, cause death.

Patients respond rapidly to treatment. In a day or so the appetite and disposition return to normal, and unless epiphyseal separation has occurred, the pain from the limbs rapidly subsides. There are no new haemorrhages and any haematuria quickly clears up. Fever, if present, is gone in two or three days. Following treatment malnutrition may be a problem; transfusion is of value in these cases. All bone lesions are rapidly cured and general improvement is usually continuous. Relapses, if they occur, are usually due to acute digestive disturbances.

LEADING MEDICAL TEXTBOOKS

Arey's Developmental Anatomy.
Beckman's Treatment.
Boyd's Pathology of Internal Diseases.
Boyd's Surgical Pathology.
Boyd's Textbook of Pathology.
Cecil's Medicine.
Christopher's Minor Surgery.
Christopher's Textbook of Surgery.
Cunningham's Manuals Practical Anatomy.
Cunningham's Textbook of Anatomy.
Curtis' Textbook of Gynaecology.
DeLee's Textbook of Obstetrics.
Dorland's Illustrated Medical Dictionary.
Griffith & Mitchell's Pediatrics.
Henderson & Gillespie's Psychiatry.
Hutchison & Hunter's Clinical Methods.
Jordan's General Bacteriology.
Maximow & Bloom's Histology.
Meakins' Medicine.
Norris & Landis' Diseases of the Chest.
Ranson's Anatomy of the Nervous System.
Schumann's Textbook of Obstetrics.
Sollmann's Pharmacology.
Todd & Sanford's Laboratory Diagnosis.
Treves & Choyce's Surgical Applied Anatomy.
Wright's Physiology.

McAinsh & Co. Limited

Dealers in Good Books Since 1885

388 YONGE ST. TORONTO

THE PHYSIOLOGY OF THE HYPOTHALAMUS

By F. L. LAWSON, '39

"The hypothalamus has now become the happy hunting-ground of experimental anatomists and physiologists, while metabolic experts find it a haven for ephemeral thoughts." (Lancet).

Anatomical Note

The hypothalamus proper is formed from the diencephalic portion of the vegetative intermediate plate of the primitive neural tube and from that portion of the subependymal plate in relation to it. In direct continuity with this latter is the subependymal matter of the preoptic region, the thalamus and the midbrain, which also has a vegetative origin and is therefore vegetative in function. The pars optica hypothalami, including the optic chiasma and the supraoptic commissures of Gudden, Meynert and Ganser, is of telencephalic origin.

Grossly the hypothalamus extends from the level of the rostral border of the optic chiasma to the caudal borders of the mammillary bodies. It blends imperceptibly with the preoptic region rostrally, the subthalamus dorsally and the tegmentum of the midbrain dorsocaudally. Developmentally it includes the pars nervosa hypophysis and in certain lower forms the saccus vasculosus, an organ for the perception of intracerebral fluid pressure.

The hypothalamus may be divided into two major complexes, four nuclear regions and six principle cell type nuclei. The supraoptic and tuberal regions are in the anterior hypothalamus, and the posterior and mammillary regions in the posterior hypothalamus. The *nucleus paraventricularis* is a large-celled group in the supraoptic region, directed vertically above the optic chiasma in close relation to the ventricle. The *nucleus supraopticus,* phylogenetically related to the latter through a common progenitor, the nucleus preopticus magnocellularis, is more laterally situated in the same region, in relation to the optic chiasma and tract. The *substantia grisea* is represented in all the regions except the mammillary but is most concentrated in the tuberal region, directly above the infundibulum, where it forms a condensation known as the nucleus hypothalamicus ventromedialis or principalis of Ramon y Cajal. Other substantia grisea cell groups are the nuclei hypothalamicus anterior, ovoideus, supraopticus diffusus and hypothalamicus parvocellularis in the supraoptic region, hypothalamicus dorsomedialis, hypothalamicus ventrolateralis and infundibularis in the tuberal region, hypothalamicus posterior, premamillaris and supramamillaris in the posterior region, perifornicalis in relation to the fornix, and periventricularis covering the ventricular wall. The lateral hypothalamic area forms the lateral portion of the hypothalamus and the dorsal hypothalamic area, limits it above. The *nucleus tuberis lateralis* forms three small macroscopic condensations in the lateral part of the tuber, i.e. in the lateral hypothalamic area in man and is more diffusely represented in the same region in lower animals. A nucleus pallidoinfundibularis, not of substantia grisea, is situated in the tuberal region between the nucleus tuberis lateralis and the optic tract. The *nucleus tuberomamillaris* is composed of large cells diffusely scattered amongst the substantia grisea cells of the posterior region and more concentrated about the mammillary nuclei, the fornix and the mammillothalamic tract, and extends forwards in the lateral hypothalamic area in relation to the nucleus tuberis lateralis. Each *corpus mamillare* includes a medial and a lateral mammillary nucleus and a nucleus intercalatus.

The hypothalamus has afferent and efferent connections with the cortex, the

extrapyramidal centres, the thalamus, probably the cerebellum, the lower brian stem and the spinal cord. So profuse are these, in adition to intrinsic connections, that Krieg has said: "In view of our still meagre knowledge of the fiber connections of these regions, it is impossible to decide whether the schema presented for the armadillo, rat, cat, dog, and to a less extent, monkey, represents an analysis of genuine anatomical and functional importance, or is to a large extent an unconscious projection of the desire to find a complicated morphology paralleling the unbelievable diversity of function at present attributed to this region." Included in the pathways conducting fibres to the hypothalamus are the fornix, the medical corticohypothalamic tract, the medial forebrain bundle, the stria terminalis, the periventricular system, the thalamic peduncles, the ansae lenticularis and peduncularis, the fasciculus tuberis descendens, the mammillary peduncle and the medial longitudinal bundle. Amongst the efferent pathways are the hypothalamo-hypophyseal tract from all the nuclei of the anterior hypothalamus, the mammillothalamic tract, the mammillotegmental tract and dorsal longitudinal bundle of Schutz, and the periventricular system and tractus hypothalamicus descendens from all the hypothalamic nuclei.

Hypophyseal secretions can act on the hypothalamus by passing into the systemic circulation, into the hypophyseoportal system, up the Herring spaces and perivascular channels to the hypothalamic cells, and into the cerebrospinal fluid (in the dog). The direct infundibular nerve connections, to partes nervosa intermedia, tuberalis and possibly anterior hypophysis, and the innervation through the thoracic autonomic system, of partes anterior and possibly tuberalis, complete the integration of the hypothalamohypophyseal mechanism.

Discussion of the Physiology

"This small portion of the nervous system is being credited with the control of multitudinous functions related to the internal economy of the human frame, and though there is undoubtedly good evidence that it controls some functions, it would be well to halt and consider carefully before attributing any more to it." (Lancet).

WATER AND CHLORIDE METABOLISM

Diabetes insipidus is a condition characterized by polyuria, polydipsia, hyperchloraemia and hypochloruria. It appears from recent work on the nervous control of water metabolism in the body that a permanent condition of diabetes insipidus can occur only in the absence of functional posterior lobe hypophyseal tissue, or other tissue capable of secreting the antidiuretic factor normally produced in the posterior lobe, and in the presence of functional anterior lobe hypophyseal tissue. The anterior lobe secretes a diuretic factor which is necessary to the occurrence of the polyuria. The factor is not the thyrotropic hormone, but is active only in the presence of thyroid, according to White and Heinbecker. The antidiuretic factor of the posterior lobe is probably secreted by the pars intermedia, and possibly by the pars tuberalis. This secretion is controlled by the supraoptic nuclei of the hypothalamus, but may possibly occur in the absence of such control. The site of action of the antidiuretic factor is uncertain since good evidence points both to a direct action on the kidneys and to an action through tuberal hypothalamic centres.

Bilateral injury to the supraopticohypophyseal system, as performed by Fisher, Ingram and Ranson, results frequently in the occurrence of a transient polyuria, perhaps due to irritation of the anterior lobe or to cessation of secretion of the antidiuretic factor, followed in a few days by a return to normal water exchange. However, within a few weeks the pars nervosa atrophies and the polyuria becomes permanent. Farr, Hare and Phillips have observed that such animals are extremely sensitive to the diuretic action of injections of anterior hypophysis.

According to Mahoney and Sheehan clipping the infundibulum in the dog causes a diabetes insipidus abolished by thyroidectomy. They find that such clipping does not result in polyuria in the monkey, however, and attribute this fact to an anatomical difference in the infundibulum in the two species. In the dog there results extensive degeneration of practically the whole hypophysis, which in the monkey does not occur, the pars tuberalis in the latter also remaining in connection with the tuber cinereum. A further lesion in the hypothalamus of the monkey now precipitates polyuria. Mahoney finds that the totally hypophysectomized chimpanzee does not show signs of polyuria or polydipsia, which is reasonable since all anterior lobe tissue is removed. Fisher, Ingram and Ranson mention that the antidiuretic factor is active in the animal with denervated kidneys, sectioned splanchnic nerves, sectioned vagi, sectioned cervical cord and sectioned brain stem, which rather indicates that the factor acts directly on the kidneys. However, Biggart draws attention to the fact that pituitrin was not active in a case of diabetes insipidus in which the tuber nuclei, in addition to the supraoptic nuclei, were destroyed. This is of special interest since it is known that pituitrin has no antidiuretic action in decerebrate, sleeping or deeply narcotized animals. The correlation of these facts is not yet possible, but it may be that the antidiuretic factor inhibits, through the tuber nuclei, the secretion of anterior lobe diuretic factor or thyroid activity, thus limiting production of that factor necessary to the action of the above and also preventing excessive breakdown of metabolites, so that normal osmotic relations are maintained in the body fluids.

CARBOHYDRATE METABOLISM

Donhoffer and Macleod showed in 1932 that decerebration or injury in the region of the hypothalamus in rabbits under luminal or amytal anaesthesia only occasionally causes the blood sugar to rise, whereas decerebration involving the pons or a narrow portion of the medulla adjacent to it causes marked hyperglycaemia within two hours. They believe that the hyperglycaemia is a result of irritation of a centre, probably in the tegmentum of the pons, and is not due to a destruction of nerve cells or pathways.

However, there is considerable evidence supporting the view that the hypothalamus plays some part in the regulation of the sugar concentration of the blood. According to Lewy and Gassman unilateral stimulation with subsequent destruction of the paraventricular nucleus causes transient hyperglycaemia. Barris and Ingram have found that bilateral lesions of the paraventricular nuclei cause chronic hypoglycaemia, frequently preceded by transient hyperglycaemia. In addition cell degeneration occurs in the paraventricular nuclei in cases of diabetes mellitus (Morgan, Vonderahe and Malone), following pancreatectomy and after piqure into the dorsal vagus nucleus. The latter is of interest since the vagus is believed to carry fibres concerned in the regulation of carbohydrate metabolism. Beattie, Brow and Long report that hyperglycaemia and glycosuria result from section of descending hypothalamic pathways caudal to the mammillary bodies and Beattie cites that small lesions of the tuber produce similar conditions of the blood and urine. Stimulation of the retrohypophysary portion of the floor of the third ventricle has been shown by Jaegher and Van Bogaert to cause hyperglycaemia, abolished and even converted into a slight hypoglycaemia by suprarenalectomy.

The British Medical Journal points out that it might be interpreted from recent work on the relations of the hypothalamus, the hypophysis and the superior cervical sympathetic ganglion in carbohydrate metabolism that, instead of active principles, inactive precursors of these may be elaborated in

the hypophyseal cells under the control of the hypothalamus or other endocrine glands, and that these may not be activated and thrown into the circulation until the necessary stimulus arrives over the superior cervical autonomic pathway. Probably this reference is to the work of Davis, Cleveland and Ingram, who found that, on condition that glycogen was available in the liver, stimulation of the superior cervical ganglion caused hyperglycaemia and glycosuria. (These effects can also be produced by direct stimulation of the hypophysis, unless the posterior lobe is removed, and are not prevented by section of the vagi or of the cord at the 4th thoracic segment). The results were not obtained, however, if the splanchnics were cut or if bilateral lesions were made in the perifornical regions of the tuber cinereum, at the level of and involving the ventromedial (substantia grisea) hypothalamic nuclei, nor were they produced by stimulation of the superior cervical sympathetic trunk. Stimulation of the stellate ganglion caused an even greater degree of hyperglycaemia and glycosuria and this was diminished but not abolished by section of the sympathetic trunk either above or below this ganglion. They also reaffirmed the observation that pancreatectomy is not followed by hyperglycaemia and glycosuria after lesions of the hypophysis and showed that this is also the case if, instead of in the hypophysis, bilateral lesions are made in the tuber cinereum in the fornical and ventromedial regions. However, it has been shown that the diabetogenic factor of the anterior hypophysis is effective in the absence of the pancreas, hypophysis, thyroid, suprarenals, ovaries, testes, splanchnic nerves, lumbar sympathetic chains or with a lesion of the tuber cinereum, the liver alone being essential.

It may be assumed that a hypothalamic lesion acts on sugar metabolism through the nervous system and the hypophysis; through the former on the suprarenals and probably the pancreatic islets (Beattie) and liver; through the latter (a) by injury to the site of action of certain of the hormones of the anterior hypophysis, since it has been shown that lesions depress diabetogenic activity of the hormones and ameliorate pancreatic diabetes, and (b) by direct depressant influence on the anterior hypophysis, which maintains the suprarenal cortex, and on the posterior hypophysis, the action of which is dependent on the liver and suprarenals. Under normal conditions it is probable that the cerebral blood sugar determines the level of blood sugar in the body generally.

FAT METABOLISM

It is evident from clinical and experimental studies that the hypothalamohypophyseal mechanism is in some way concerned in the regulation of body fat. Beattie quotes Raab as considering that the posterior hypophysis and tuber nuclei influence the metabolism of fat in the liver since, when efferent paths from the tuber are destroyed or the hypophysis is destroyed, there is an increased deposition of fat in the body. Keller and Noble have observed an enhanced appetite, with resultant increase in body weight due to fat deposition, in dogs in which the pars intermedia-infundibular complex had been successfully removed. However, adiposity never occurred under any conditions with total anterior lobectomy. Mahoney and Sheehan found that clipping the infundibulum in dogs caused a condition of adiposity and genital atrophy, similar to the Fröhlich syndrome, accompanying diabetes insipidus. This did not occur when the same procedure was carried out in monkeys. They attributed their results to a species difference in the anatomy of the infundibular region, in the dog the hypothalamus and hypophysis being more intimately linked vascularly and nervously. Lhermitte, in his review, attributes diencephalic obesity, progressive lipodystrophy and the lipodystrophy associated with the Laur-

ence-Moon-Biedl syndrome to hypothalamic pathology but, although several workers have produced the adiposogenital syndrome of Fröhlich in animals by tuberal lesions, Ranson, from a large series of cats with extensive lesions in various parts of the tuber cinereum, has accumulated only negative evidence. His animals showed neither adiposity nor impairment of genital function and raised normal families.

Van Bogaert and Van Meel have demonstrated a fall of 40% in lipaemia on stimulation of the retrohypophysary portion of the floor of the third ventricle, the change being almost entirely due to fatty acids. This is of interest since the cells of the tuberomammillary nucleus, located in the posterior hypothalamus, are charged with lipoidal substance even in health.

Probably the hypothalamus influences protein metabolism also, since Houssay has observed that the absolute augmentation of metabolism produced by a meat meal is similar in hypophysectomized and normal dogs, but is much greater in dogs with lesions of the tuber cinereum.

THE CIRCULATORY SYSTEM

In practically all the experimental physiological studies on the hypothalamus some effect on the circulatory system has been produced and most commonly this effect has been of a sympathetic or pressor nature. Beattie, Brow and Long found that stimulation of the lateral wall of the third ventricle between the tuberal region and the opening of the aqueduct into the third ventricle, i.e. in the region of the posterior and supramammillary nuclei, caused an increased heart rate and diminished AV conduction time, accompanied by epinephrin secretion and other sympathetic phenomena. Stimulation under chloroform caused ventricular extrasystoles. On section caudal to this part of the brain the AV conduction time was increased and the cardiac extrasystolic arrhythmia associated with the chloroform anaesthesia was abolished. Ranson, Kabat and Magoun, and Crouch and Elliott, by stimulating the lateral hypothalamic area have produced a blood pressure rise, accompanied by other sympathetic effects. Increases in both blood pressure and heart rate have been observed by Morgan following lesions of the tuber cinereum in the dog. Van Bogaert and his associates have stimulated the retrohypophysary portion of the hypothalamus in the region of the mammillary bodies with resultant generalized intense convulsions, emission of urine, marked respiratory changes, and a great increase in arterial blood pressure accompanied by tachycardia. These effects were coextensive and proportional to the stimulation and were frequently followed by a marked bradycardia, which did not occur if the vagi were cut in the neck. By appropriate means they were able to demonstrate that the circulatory effects were independent of the convulsions and respiratory changes. Dikshit, by intraventricular injections of acetylcholine, nicotine or caffeine, caused cardiac irregularities due to excitation of the hypothalamic centres. Much smaller amounts of caffeine than in the intravenous minimum were effective. Watts and Fulton have found that lesions in the hypothalamus and especially of the supraoptic and tuber (substantia grisea) nuclei in monkeys cause cardiac irregularities, including extrasystoles, with a tendency towards death from intracardiac clotting, possibly due to fibrillation resulting from a release or irritation of the sympathetic complex. It is, therefore, doubly of interest that Page has noted a great similarity between diencephalic sympathetic circulatory effects and the phenomena observed in a syndrome occuring in young people with essential hypertension.

Somewhat less frequently parasympathetic or depressor effects have been obtained from the hypothalamus.

Van Bogaert has observed a primary bradycardia, abolished by section of the vagi and followed by a tachycardial phase, on stimulation of the infundibulotuberine region, in addition to that strong vagal bradycardia following the return of tachycardia to normal after retrohypophysary stimulation. Beattie, also, has caused a slowing of the heart by stimulating the tuberal region. In addition, Van Bogaert has obtained durable and consistent hypotension with slight bradycardia on stimulation of the laterohypophysary region, just at the bifurcation of the internal carotid artery or the junction of the hippocampus with the floor of the third ventricle. It appears, therefore, that in the hypothalamus, just as in the frontal lobe cortex, there are immediately adjacent pressor and depressor points. Ranson, however, has been able to obtain hypotension and bradycardia only from the septal region and only on occasion.

Stavraky has shown that stimulation of the dorsal or "posterior" hypothalamus causes a pial vasoconstriction with the highest threshold of any of the sympathetic responses obtained from this region. The effect is not dependent on respiratory changes, the suprarenals or the hypophysis. Stimulation of the most ventral and ventrolateral parts of the tuber cinereum causes a bilateral dilatation of the pial arteries accompanied by a lowering of the blood pressure and a slight decrease in heart rate, phenomena similar to those observed in the diving animal following inflation of the lungs. This control of the cerebral blood vessels is of interest since it is known that constriction of the cerebral blood vessels frequently precedes an epileptic attack (Keyl) and since Morgan and Johnson have demonstrated hypothalamic degeneration in cases of idiopathic epilepsy. However, an intrinsic chemical regulation, with CO^2 playing the dominant role, seems more important than nerve control of the hypothalamic vessels themselves, in the opinion of Schmidt.

THE RESPIRATORY SYSTEM

Kabat, Anson, Magoun and Ranson have observed that stimulation of the lateral hypothalamic area causes an increase in rate and depth of respiration, and according to Ranson, Kabat and Magoun these reactions can be followed caudad into the tegmentum of the midbrain and the central gray matter of the aqueduct, with evidence of some crossing in the supramammillary decussation. Stavraky caused the production of hyperpnoea, along with pupillary dilatation, nictitating membrane retraction, hypertension, piloerection, salivation, lacrimation, cries and urination, by stimulation of the dorsal or "posterior" hypothalamus. Crouch and Elliott and Ectors have shown that stimulating the supramammillary region results in rapid, shallow breathing or panting, whereas rapid, deep respiration is produced when the anterior hypothalamus is stimulated.

Pinkston, Bard and Rioch, in investigating the temperature-regulating mechanism, demonstrated in dogs with the frontal lobes removed but the hypothalamus left intact, an immediate and vigorous shivering in response to cold, but absence of true polypnoeic panting. They recall that Keller and Hare found that, after the hypothalamus was removed, leaving the rest of the brain intact, the animal could not be made to shiver but continuous polypnoea occurred. Otenasek and Lilienthal have shown that the mechanism responsible for decorticate polypnoeic panting is separate from and located in the diencephalon dorsal to the mechanism concerned in sham rage, i.e. the posterior hypothalamus, in the cat. It is suggested that there is a reciprocal mechanism for panting and shivering, and when the centre for one is removed that for the other is released.

Ranson, Kabat and Magoun found that stimulation of the gray matter surrounding the anterior commissure not far from the midline or stimulation

of the adjacent portion of the septum inhibits respiration.

THE GASTROINTESTINAL SYSTEM

Kabat, Anson, Magoun and Ranson have shown that stimulation of the lateral hypothalamic area in the unanaesthetized cat causes inhibition of gastrointestinal motility and decrease in tone, along with other evidence of a sympathetic discharge. Ranson, Kabat and Magoun found that these reactions could be followed caudad just as the respiratory responses previously described.

Beattie and Sheehan have also demonstrated similar gut inhibition by stimulation of the posterior hypothalamus but, in addition, they were able to produce parasympathetic reactions by stimulation of the tuberal region. The latter gave constantly a rise in intragastric pressure, accompanied by a fall in blood pressure and a subsequent increase in peristaltic movements of the stomach and these effects were abolished by section of the vagi. Stimulation of the lateral margin of the infundibulum has produced hypermotility, hypersecretion and hyperaemia of the stomach. Cushing caused similar results by injection of polocarpine or posterior hypophyseal extract, but not pitocin or pitressin, into the third ventricle. Watts and Fulton attribute profound intestinal disturbances, with gastric and duodenal erosions, haemorrhages and even perforation, following lesions involving the supraoptic and tuber nuclei, to a release or irritation of the sympathetic mechanism of the gut.

Keller, and Keller and D'Amour, have studied the relation of the hypothalamus to gastrointestinal lesions at greater length. Two chief gastric effects produced were spontaneous haemorrhagic states and crater formation. They conclude that the haemorrhagic states are produced through the sympathetic nerves by activation of the sympathetic or posterior hypothalamic mechanism, and the craters through the vagi by activation of the parasympathetic or anterior hypothalamic mechanism. It is probable that, although vasomotor disturbances occur in both conditions, the primary derangement is in the secretory processes.

Editor's Note: This is the first of two papers on the physiology of the hypothalamus. The remainder of the physiology will be discussed by the author in another paper to appear shortly.

NOTICE

Would the person or persons responsible for borrowing the large silver 'Assault at Arms' cup from the Medical Office last March (1937) please return same as soon as possible.

The J. F. HARTZ CO., Limited

Carry a complete line of

MEDICAL STUDENTS' SUPPLIES

STETHOSCOPES, OTOLARYNGEAL SETS,
HAEMOCYTOMETERS, HAEMOGLOBINOMETERS.
STUDENTS' DISSECTING SETS

34 GRENVILLE STREET TORONTO, CANADA

Feature Section

THE PATH TO SUCCESS.

Perhaps 'The Path to Success' might be an appropriate general title for the series of biographies which we hope to publish in these columns. In any case, we have always been interested in learning of the paths by which our esteemed Teachers have ascended to their present eminences. Besides, we want to know our Teachers better. They have often seemed remote, untouchable, almost superhuman. If we could only learn of their beginnings, their school life, their post-graduate appointments, their early struggles in the practice of medicine and finally of the steps by which they have attained their present degree of success, then surely they would seem more human, even like ourselves. Surely we would be inspired by their precept and, as we discerned the qualities which have served them so well we might be encouraged to develop those qualities in ourselves. Thus we hope that our readers will find these biographies both interesting and instructive.

We have pleasure in presenting as the first in our series a biography of our former Dean of the Faculty of Medicine and present Professor of Hygiene and Preventive Medicine, Dr. J. G. FitzGerald.

J. G. FITZGERALD, M.B., M.D. (Tor.), LL.D., F.R.S.C., F.R.C.P. (C.).

John Gerald FitzGerald was born in Drayton, Ontario, in 1882, received his preliminary education in Harriston and graduated from the Faculty of Medicine, University of Toronto, in 1903, the youngest member of his class.

After several years spent in psychiatry and neuropathology in Toronto, at the Harvard Medical School in Baltimore and in other centres, Dr. FitzGerald's interest turned to bacteriology. In 1909 he was appointed Lecturer in Bacteriology in the University of Toronto, and he spent the next two years doing research work at the Pasteur Institute in Paris and Brussels and at the University of Frieburg.

From 1911 to 1913 he was Associate Professor of Bacteriology in the University of California and in 1913 was appointed Associate Professor of Hygiene in the University of Toronto, and in 1914 Director of the Antitoxin Laboratory in the Department of Hygiene.

During the Great War, Dr. FitzGerald served as a Captain in the C.A.M.C. and later as a Major in the R.A.M.C. of the British Expeditionary Force, being officer in charge of a Mobile Laboratory in France and Assistant Advisor in Pathology, Fifth British Army.

In 1919 he was made Professor of Hygiene and Preventive Medicine in the University of Toronto and director of the Connaught Laboratories where antitoxins, sera, vaccines, insulin and other products are made and investigated. In 1922-1923 he occupied the Chair of Bacteriology and Experimental Pathology in the University of California.

In 1923 Professor FitzGerald was made a member of the International Board of Health, which has recently become a division of the Rockefeller Foundation and of which division he is a Scientific Director. Chiefly through his efforts, the Foundation made a grant in 1925 for the establishment of the School of Hygiene at Toronto and a further grant in 1930 for its extension. Professor FitzGerald was made Director of the School on its foundation and continues to direct investigations be-

ing carried on there in the fields of Public Health, Hygiene and Preventive Medicine. On these subjects he has written widely and is a recognized authority.

His appointment in 1930 to the Health Committee of the League of Nations is a mark of the world-wide reputation which he has attained. The following list testifies to the extent of his activities:

Fellow of the Royal Society of Canada and Member of Council.

Fellow of the American Public Health Association.

F.R.C.P. and S. (Canada) and Member of Council.

Member of the Dominion Council of Health.

Trustee of the Banting Research Foundation.

Member of the Executives of the Canadian Medical Association, Canadian Public Health Association and Canadian Tuberculosis Association.

Member of the Advisory and Research Committee of the Canadian National Committee of Mental Hygiene.

Honorary Advisor in Public Health Administration, Department of Health, Ontario.

Member Advisory Council of the Ontario Research Foundation.

Member of Associate Committee on Tuberculosis Research of the National Research Council of Canada.

Fellow of the Academy of Medicine, Toronto.

On January the first, 1932, Professor FitzGerald became Dean of the Faculty of Medicine to succeed Dr. Primrose, who had occupied the position since 1920. During his tenure of office Dean FitzGerald spared no energies in understanding and solving the problems of staff and student. His never-failing courtesy, tact, and good humour made him popular with everyone who knew him. It was with regret that we learned last year of his resignation from the Deanship.

Dr. FitzGerald during the past year embarked upon an extensive survey of the methods of teaching preventive medicine in medical schools throughout Canada, the United States, Great Britain and Europe. This investigation was conducted on behalf of the International Health Division of the Rockefeller Foundation of which Division he continues as a Scientific Director. He has now resumed his responsibilities as Director of the Connaught Laboratories and of the School of Hygiene.

A charming personality, undoubted scholastic ability and conscientious hard work have ever combined to make his path to success both swift and sure.

The Editor wishes to thank Dr. C. H. Best for kind assistance in preparing the above account.

NEUTRALITY REGULATION OF THE ORGANISM.

The medical faculty and students enjoyed on October the fifteenth the privilege of seeing and listening to one of the outstanding scientists of our time. The speaker was Dr. D. D. Van Slyke of the Rockefeller Hospital. New York, who, for his outstanding contributions to the knowledge of blood chemistry, was awarded the Charles Mickle Fellowship for 1936. This Fellowship is presented by the University of Toronto to that member of the medical profession who is considered by the council of the Faculty of Medicine to have done most during the preceding ten years to advance sound knowledge of a practical kind in medical art or science.

Dr. Van Slyke traced in his lecture the sequence of his own researches and the advancing knowledge of Neutrality Regulation which resulted therefrom. He told how his interest in the subject began twenty years ago when, as a newly appointed member of the staff of the Rockefeller Hospital, he was presented with the problem of detecting the onset of diabetic acidosis be-

fore the development of coma. It became obvious that a simple and accurate method of measuring blood bicarbonate was required and the well known Van Slyke apparatus for this purpose was soon devised. This method, in which the volume of the carbon dioxide released was measured at known pressure, was shortly replaced by a manometric method in which was measured the pressure required to reduce the gas to a constant volume. This method proved much more accurate than its predecessor and from it has been evolved methods for measuring any blood constituent which will release a gas when treated with the appropriate reagents A colorimetric method for determining pH of blood using methyl red was described and particularly interesting was the description of an apparatus developed by Hastings in which both pH and total carbon dioxide content can be measured using only 0.1cc. of plasma.

Dr. Van Slyke went on to discuss the modern conception of neutrality regulation in the human body. He explained in an interesting manner the method by which the "chloride shift" enables the blood cells to assist in the stabilization of the plasma pH and pointed out that the method by which the tissue cells assist in maintaining the neutrality of the interstitial fluid is as yet unknown. The rôle of the kidney in acid-base regulation was discussed and it was shown that the kidney is capable within certain limits of excreting a mixture containing varying ratios of carbonic acid to bicarbonate. A factor frequently overlooked in considering the defences of the body against acid is the release of free base by the combustion of organic acids.

One's general impression of the lecture was best expressed by the words which Dr. Hunter used in moving a vote of thanks to Dr. Van Slyke, "There is no pleasure greater than that of listening to a complex subject expounded by one who is a master of that subject and who has himself contributed much of the knowledge of that subject."

FIFTEEN GREATEST PHYSICIANS OF ALL TIMES.

The Fifteen Greatest Physicians of All Times are named in a past issue of "Hygeia." The names are taken from a list recently made by medical historians, in an effort to name the five greatest contributors to the advancement of medicine.

Some of these men are not really physicians, in the sense that they have practised medicine or have obtained a medical degree. Their contributions are in fields closely related to medicine. "Hygeia's" list is as follows:

1. Hippocrates, father of modern medicine, whose school and writings formulated our knowledge of medicine.
2. Galen, who described the action of the heart valves, elevated the status of the medical profession and collected the knowledge of his time in a system of medicine which was followed by physicians for centuries.
3. Leonardo de Vinci, artist and inventor, who contributed greatly to the science of anatomy.
4. Vesalius, father of modern anatomy.
5. William Harvey representative of the renaissance of medicine, who first described scientifically the circulation of the blood.
6. Sydenham, the greatest figure in medicine of the 17th century, who wrote excellent descriptions of several disease conditions.
7. John Hunter, father of surgery.
8. Auenbrugger, who first described the method of tapping the chest for the determination of conditions beneath the skin.
9. Laennec, who first described the use of the stethoscope for listening to sounds within the chest.
10. Pasteur, father of the conception of bacteria as the direct cause of many diseases.
11. Robert Koch, discoverer of the tubercle bacillus, who established bacteriology as a science.
12. Lister, who showed how bacterial infection may be prevented.

13, 14, 15. James Young Simpson, William Morton and Crawford Long, who first made use of anesthetics and made surgery painless.

DR. E. S. RYERSON, M.D., C.M.

An honour of which most students are unaware has been bestowed during the past year upon our assistant Dean, Dr. Ryerson. He was elected President of the Association of American Medical Colleges for the term 1936-37 and as such is giving the Presidential address at the meeting in San Francisco in October. This society is composed of representatives from the Faculties of all the Medical Colleges of North America and is interested mainly in the problem of medical education. As a member of the Association Dr. Ryerson accompanied by Dr. Weiskoten, Dean and Professor of Pathology, University of Syracuse, made an inspection of the Medical Schools of Wayne University and the University of Michigan in November, 1935, and of the University of Rochester in May, 1936. More recently with Dr. W. D. Cutter, Secretary of the Council on Medical Education and Hospitals of the American Medical Association, he made a survey of the Universities of Alberta, Saskatchewan and Manitoba in April, 1936

It is noteworthy that through these activities of Dr. Ryerson, who is a well-recognized authority on medical education, our university is attaining a valuable prestige among medical authorities throughout North America.

AN INTERESTING SUMMER.

By R. Alexander Mustard, '38.

I consider myself very fortunate to have spent July and August of this year as doctor in a boys' camp. I had been a little doubtful about accepting the position that was offered to me. There wasn't much money involved. But I felt that I needed a holiday, so I took my chance to get one at somebody else's expense. Certainly I shall never regret it. The camp was situated on one of the delightful little lakes which dot Haliburton County and the surrounding country was most beautiful. It did not take long for the carefree life in camp, the swimming, canoeing, fishing, hiking and sunbathing to eradicate the ravishes of a hectic school year and substitute a healthy coat of tan. Food never tasted so good nor sleep so restful. The exultant glow of physical fitness, which had long been forgotten, was experienced once again and my return to the city found me ready to conquer the world. I think that every medical student should have such a holiday at the end of his fifth year because probably never again will he have the opportunity for a vacation so long and so free from care.

I soon found that looking after a group of boys living together in a camp was really a problem in public health. Prevention of illness was of major importance. In this respect, one had first to think of the communicable diseases. Every boy was carefully examined on admittance to the camp and questioned closely regarding contact with measles, whooping cough, scarlet fever, etc Then each boy was examined at weekly intervals and a record kept of his weight and general condition. In this connection it is perhaps interesting to note that I have found it far harder to be sure that a person is in normal good health than it has ever been to detect the signs of disease in one who is obviously ill. Unfortunately, despite all my watchfulness, I one day found myself with a full-fledged case of "pink eye" in one of my charges. I had the boy promptly isolated as far as is possible in a camp and allowed him no swimming. I was optimistic enough to think I had nipped an epidemic in the bud. One can imagine my chagrin as case after case developed despite all my efforts. "Pink eye" haunted me the whole summer and I don't think a day went by when I didn't have to treat someone's eyes with boracic solution and ointment. It

was positively fiendish the way practically every boy would develop the condition just at the time when his parents were coming to visit him. Fortunately, the withering scorn, which these fond parents undoubtedly felt for the doctor who would let their son develop such a ghastly condition, was not often expressed in words. Pink-eye was for me a lesson in humility.

I found that the knowledge of hygiene acquired in the field course just a week or so before camp opened proved to be of unquestionable value. On being consulted about the food supply for the camp I was able to explain why only meat stamped "Canada Approved" should be bought and why, since pasteurized milk was not available, we should insist on getting the camp supply from tuberculin-tested cows. I was able to examine the well from which the drinking water was obtained and to criticize its construction and make recommendations for its improvement in a way which the camp directors found quite impressive. My prestige was further established by my knowing exactly how to go about chlorinating the water so as to render it safe for drinking. The construction and care of a sanitary privvy was further knowledge which proved to be very useful.

I thought when I went away to camp that I knew quite a bit about the practice of medicine but I soon found that there is a world of difference between theory and practice. I knew, or thought I knew, all about bronchiectasis and empyema, about duodenal ulcer and intestinal obstruction, about hemiplegia and arteriosclerosis and about a lot of other things that I never saw in my limited practice. Instead of these major diseases I found myself confronted with a whole host of little things of which I never thought much before. Sunburn and sick stomachs, diarrhoea and "belly aches" were my constant problems and I found my knowledge of what to do rather vague. I was quite perturbed, if not a little disappointed, to find that all abdominal pains did not mean appendicitis. In fact I didn't find a case of appendicitis all summer. Bee stings were an emergency with which I had not reckoned and I searched in vain through my text books of medicine and surgery for a clue as to their treatment. When I saw a mother, whose child had been stung, put moist baking soda on the lesions and obtain considerable relief, it occurred to me that the whole of medical science was not taught in the classroom. Then there was that peculiar punctate rash which I observed on the backs and arms of so many boys during the first few weeks of camp life. It didn't resemble any of the skin diseases with which I was familiar. Enlightenment came when one boy told me of course he got it every year and did I not know prickly heat when I saw it? In my course in surgery I had learnt all about dressing a wound but it had never occurred to me that I did not even know how to bandage a finger or a hand or a knee. Of course I managed to cover them up and to keep them covered by the utilization of much adhesive tape and indeed, I eventually developed a decent technique of bandaging but I certainly was sorry that I had never learnt that simple procedure before. Towards the end of the summer I obtained a supply of "Band-Aids" which cut my work just about in half.

Fortunately, a few things developed which I was competent to treat. They were simple lesions simply treated but they showed me how easily a doctor's reputation can be established by such trifles. A special pair of tweezers which would seize and extract even the tiniest sliver brought me fame which I was loathe to distain. A bottle of Keith's dressing which stopped earache as if by magic, a supply of "Dynamite pills" in the form of "A.B.S. & C.'s," which never failed to produce results, magnesium sulphite-glycerin paste for boils,—these established me as a wonder worker.

Only one boy developed a serious illness during the summer. It turned out to be a lobar pneumonia but it was

not possible to establish the diagnosis definitely until sixteen hours had elapsed since the onset of malaise. During that time I was able to observe how difficult it may be to distinguish an early pneumonia from an intra-abdominal lesion, or in this case, from a pyelitis. And it was impressed upon me, because I saw it for myself, that the earliest signs in the chest are an increased respiratory rate and decreased breath sounds.

One Sunday afternoon I had a rather exciting time. We were eating lunch in the dining hall when a severe electric storm developed. Suddenly a terrific crash and a blinding flash of light came from the open door nearby. Several women, visitors to the camp, screamed. Three of them had apparently received part of the bolt through their bodies. They, of course, promptly fainted and recovered shortly only to pass into a state which might be described as "hysterical." One woman in particular seemed to be seriously affected. She had marked dyspnoea of the gasping type and her pulse was rapid and very feeble. I was considerably alarmed at the fate which had thrust so grave a case into my untrained hands. Fortunately whiskey and coramine were available and the combined effect of these stimulants was quite remarkable. Within a few hours the patient seemed fully recovered. The doctor, alas, didn't recover his *sang froid* until the following day and even then looked in the mirror expecting to find a few gray hairs.

Not the least interesting feature of camp life was the opportunity afforded for observing child psychology at first hand. All the types of child personality were represented, the spoiled, the repressed, the perverted, the shy child, the friendly child, the saucy child and the polite child. There were even a few which might be called "normal" in their behaviour. And it was most instructive to observe, when the parents visited, how closely the children resembled their parents in their personalities and also, how accurately their reaction-tendencies reflected the parents' treatment of them. One could guess fairly correctly what the parents would be like by studying the child's behaviour. And it seemed to me that if one wanted to improve the child's behaviour one would first have to re-educate the parents. Incidentally, I was greatly impressed by the almost universal improvement in personality and behaviour of the children brought about by a stay in camp. That type of impersonal discipline which is peculiar to camp life plus the fact that a boy is more or less on his own resources seems to have a most beneficial effect upon all.

The summer passed all too quickly. As I have pointed out already, it left me humbler, wiser and healthier. A more valuable triad could scarcely be found.

CONTRIBUTIONS.

The feature editor will be only too pleased to receive any contributions to this section of the Journal. Anecdotes of a medical flavour, interesting case reports, original poems, descriptions of interesting summer travels or occupations, etc., will all prove acceptable. Here is your chance to discover in yourself a literary genius which you may not previously have been aware that you possessed.

SUBSCRIBERS ARE RESPECTFULLY REQUESTED TO PATRONIZE FIRMS ADVERTISING IN THE JOURNAL.

Medical Society Notes

By W. K. KERR

1937-38 Medical Society Executive

President—E. B. Tovee.
Vice-President— C. C. Gray.
Secretary-Treasurer—W. K. Kerr.
Assistant Secretary-Treasurer—W. E. Ortved
President Sixth Year—W. G. Bigelow.
President Fifth Year—T. C. Jewell.
President Fourth Year—W. L. C. McQill.
President Third Year—T. H. Black.
President Second Year—D. D. Gain
President First Year—J. C. Decker.
Editor in Chief Medical Journal— W. G. Bigelow.
Chairman Daffydil Committee—T. H. Baillie.
President of M.W.U.A —Mary Albertson.
President Medical Athletic Association—W. B. Charles.

The Medical Society takes pleasure in welcoming those brave students who have enrolled in the first year of Medicine. We shall attempt in the next few months to bring you in association with students of the higher years, in order that you may learn to know some of them and derive the benefits of their past experience.

Last spring the executive discussed the desirability of bringing to the attention of the faculty the opinions of the student body on the arrangement of the curriculum of this medical school. The year presidents conducted open meetings, some of them did, and constructive criticism and suggestions were invited from students of all years. These opinions and thoughts, being in written form, were then turned over to a committee appointed by the Society and consisting of Mr. King, the chairman, and Messrs. Hayward, Breslin, Kerr and Crossland. The committee then made a report, copies of which were sent to the faculty members engaged in revising the course.

Now that we see the revised course, it appears that a number of important changes have been made which conform closely with those suggested by the students themselves last spring. This may be taken to mean one of two things; that the students have made a great contribution to the improvement of their school, or that their views on the problems were so excellent that they agreed with the changes contemplated by the faculty members. A gratifying discovery whichever side one takes.

The Association of Medical Students, a recently formed American organization, sent an invitation to the medical students of the University of Toronto to attend their first convention, held at the Johns Hopkins University School of Medicine in Baltimore, April 17-18th, 1937. Mr. King, president of the Medical Athletic Association, was sent there by the Medical Society to represent this medical school. The post-convention issue of the Journal of the A.M.S. gives a full account of the aims and activities of this organization. It now rests with

the students to consider if this medical school ought to join the Association.

At the first meeting of the present executive, April 12th, 1937, it was moved that a sum of $700 be given to the Board of Governors to be used for bursaries to needy and worthy medical students, particularly those of the upper years. The University also contributes to this fund and in direct proportion to the sum saved by the Medical Society.

This year the Medical Society will attempt to make the open meetings a success comparable to last year's and also to have a more active participation of the students at these meetings. The first one will be of vital importance to the students. Dr. Hanna will give an introductory explanation to the proposed plan of health insurance of medical students; this is to be followed by an informal discussion of the subject.

INITIATIONS.

On Friday, October the eighteenth, with four score of proverbially beautiful Whitney Hall frosh and sophs in attendance, the sophomores of 4T2 attempted to administer to the freshmen of 4T3, the sacred rites of initiation into the medical faculty. Believing that the standard of initiation into the medical faculty for the past few years has been considerably below par, the sophomores attempted to remedy this; to what extent they were successful, only the frosh can tell. After the frosh had relieved themselves of five dollars (a most important feature of initiation) for which they received a beautiful tie, with a slightly reddish cast and of exquisite texture, they were lined up in simple file in the corridor leading to lecture room 138. They were then ushered into said lecture room before a very keen and breathless audience (the ventilation was terrible) in groups of ten approximately, with their coats on backwards, pants rolled up to the knees and ties very conspicuous. Once inside this sacred den, they were requested (rather forcibly) to perform divers feats. These feats took the form of blind-folded boxing, shoving "bibles" along the floor with their noses, feeding each other jam while blind-folded and stripped to the waist (the walls and floors came out on top) and several choruses singing naturally and in falsetto (Daffydil please note: several beautiful 'soprano' voices were 'discovered.') In addition several of the frosh attempted to give Epistaxis and Toronto yells with not too much success, while a dark, handsome lad and a rather buxom blond boy attempted to give their interpretation of a Robert Taylor, Mae West love scene. With parting words to the frosh to collect all the green ties possible and to guard the red, the meeting adjourned.

C.A.S.

LEPROSY CONFERENCE

"Arrangements are being made to hold the fourth International Leprosy Conference in Cairo, beginning on the 21st of March, 1938. This conference is being organized by the International Leprosy Association, and this will be the first International Conference to be arranged by this association since its inauguration in 1931. Three previous conferences of this nature have been held—at Berlin in 1897, at Bergen in 1909 and at Strassbourg in 1923.

"The Egyptian Government is inviting all countries concerned to send official delegates. In addition to these, doctors and others interested in the subject are invited to be present. Full information can be obtained from the Secretary of the International Leprosy Association, 131 Baker Street, London, W.1."

Medical Athletics

President—W. B. Charles.
Vice-President—B. Laski.
Secretary-treasurer—P. McGoey.
Third Year Rep.—C. Cowan.
Second Year Rep.—P. Statton.
First Year Rep.—A Park.
Quartermaster—D. Garrett.

It is planned that this year each edition of the Journal shall contain a short account of the athletic activities of the undergraduates of this faculty. This has not been a regular feature of the Journal in the past and although it is not intended to inaugurate a sporting page in the ordinary sense of that phrase it is felt that some account of this rather vital phase of undergraduate life would not be out of place. In addition it is hoped that such accounts of the activities of our various interfaculty teams and our more prominent athletes in intercollegiate sport will serve further to stimulate interest.

By way of introduction and for the benefit of the freshmen we might point out that the Medical Athletic Association is composed of six members the three from the senior years taking the senior offices. It is the duty of these men, who are elected by the students, to organize the activities of "Meds" in interfaculty competition. A portion of the Medical Society fee is turned over to the Medical Athletic Association and in this way the latter is able to provide the teams with the necessary equipment. A quartermaster is appointed each year whose duty it is to purchase all equipment and to look after the stores. In addition the Association awards an athletic letter which may be applied for on fulfilling any one of the following requirements:

1. Three years participation in interfaculty sport.

2. Membership on an interfaculty championship team.

3. A first "T" holder.

At the time of writing none of our interfaculty teams have as yet tasted actual competition. The soccer and football are hard at work each night and although somewhat handicapped by the delay in the opening of school we presume that the other faculties are experiencing similar difficulties. Enthusiasm is high in anticipation of another successful season in one of the most grueling of all sports—interfaculty football.

The medical representation on the senior football team while not as numerous as in some former years has not lacked in brilliancy. We feel sure that Warren Stevens must offer up a prayer of thankfulness every time they come up unhurt, for they compose the nucleus around which the team is built.

Turney Williams follows in the footsteps of his brother Al as Captain of the team this year, and it is our sincere wish that he will be able to share with him the honour of captaining an intercollegiate championship winner. Turney is again performing in his usual brilliant fashion on the line which has gained him an enviable reputation in Canadian football. Turney is a great favourite with his team-mates and his coolness in the heat of battle has a steadying effect which enhances his value as a leader. Turney is sitting for a second year on the Athletic Directorate.

Next we have Cam Gray, perhaps the finest exponent of the placement kick in Canadian football, who seems well on his way to another intercollegiate scoring championship. Cam is Varsity's real scoring threat and his consistently brilliant play has spread his name far and wide. With all his fame Cam is the most modest of heroes, the most generous of team-mates and the best of sports.

Meds can also claim the greatest outside wing in the intercollegiate and many of those who speak with authority state he has few equals in the whole country. We refer of course to Jack Holden. No one who has seen Jack stop an opponent with one of those deadly tackles or miraculously snare a

forward pass out of mid air will deny him that title. Jack is Varsity's oldest player in point of service.

Colin (Red) Brebner is the fourth medical student on the team. Red is also one of the veterans of this year's squad and although lacking some of the lustre of his fellow students his steady play and experience can always be counted on when he is called in to the game.

Although we do not wish to spoil whatever effect the above may have we feel it necessary to mention the lamentable showing of this faculty in the interfaculty track meet. When the final results were published our lone entrant had amassed the total of one-half point and the faculty of medicine stood at the bottom of the list. This is not a comment on the ability of that particular athlete for he is to be heartily commended, but it is a sad reflection on the faculty as a whole. We feel that many a grad must have raised an eyebrow in wonderment and then thought of the good old days when Meds was a power in track and field and ran off a keenly contested field day of its own each year.

One final bright spot is the appearance of Jack Tomlinson on the intercollegiate tennis team again this year. This is nothing new for Jack but we take this opportunity of wishing him the best of luck at Queen's.

In closing we would like to repeat what you have all been told, but especially for the benefit of the junior years, the desirability—almost necessity, of participation in extra-curricular activities. True, athletics are not the only means of broadening oneself, but never let it be said that you, an able bodied medical student, spent six years at the University of Toronto with all its facilities and never took part in a sport. Go out and get your first "T" by all means. That should be the aim of every student. But if you are not quite good enuogh for the University or feel, like the most of us, that our services would not be appreciated among such supermen, come on out for the faculty. You will be very welcome and there is room for everybody.

CANADIAN
LABORATORY
SUPPLIES
LIMITED

Canada's Leading Laboratory Supply House.

Headquarters in Canada for
LABORATORY APPARATUS
AND CHEMICAL REAGENTS

MONTREAL, QUE.
296 St. Paul St. West.

TORONTO-5, ONT.
Hartz Building. 32 Grenville Street

When you are Through
with "Lectures" and "Lab"

TAKE TIME
TO PLAY

Your favorite sport, and
get the greatest enjoyment
from the game with

WILSON
EQUIPMENT

Built for Dependable Service

The Harold A. Wilson
Company, Limited
299 Yonge St. Toronto

Abstracts

CONTROL OF DIABETES MELLITUS WITH PROTAMINE ZINC INSULIN IN SURGERY

By A. F. Fowler, E. H. Bensley and I. M. Rabinowitch—*C.M.A.J., Vol.* 36, *No.* 6., *June* 1937. *p.* 561.

One of the great advantages of protamine zinc insulin is the smaller number of injections necessary to control the diabetes. In order, however, that it may be applicable to surgery, protamine zinc insulin must also possess the following attributes:

1. Rapid Action—for preparation of the diabetic for emergency operations.
2. Sustained Action—
   (a) to combat possible post-operative disturbances of carbohydrate metabolism due to trauma or anaesthesia.
   (b) to permit the use of intravenous injections of glucose whenever necessary.
   (c) to prevent delayed healing of tissue.

Cases are cited which show that even in the presence of severe infections, the blood sugar may be made to decrease as rapidly with protamine zinc insulin as with unmodified insulin, providing the dose is large (usually 100-200 units). When such large doses are given it is the writer's practice to inject the suspension at three or four different sites of the body, not only to avoid the discomfort of the distention of the tissues due to bulk but also to insure more rapid absorption.

In order to demonstrate the sustained action of protamine zinc insulin in patients with surgical conditions, two strictly comparable series of cases undergoing similar operations were selected. One series was carried through the operation with unmodified insulin received two to four times a day, while the other frogs went through the pre-operative, operative and post-operative stages with only one dose of protamine zinc insulin per day. Blood and urine sugar determinations revealed that the control of the diabetes both before and after the operation was much better with the protamine zinc insulin than with the unmodified insulin.

To illustrate the feasibility of administering post-operatively intravenous glucose to diabetic patients receiving protamine zinc insulin, a case is cited of a severe diabetic undergoing a diolecystectomy, who received 750cc. of 5% glucose intravenously the day following operation and utilized it completely as indicated by the failure of sugar to appear in the urine.

In regard to the effect of controlling the diabetes by protamine zinc insulin upon the healing of wounds, it is pointed out that in a large number of cases with no infection the number of days after operation when (a) the clips were removed; when (b) the sutures were removed; when (c) the patients were allowed out of bed; and when (d) they were discharged from hospital did not differ from the time required for non-diabetics with similar surgical conditions and exposed to similar types of operations. The efficiency of protamine zinc insulin in promoting healing in the presence of infection is well illustrated by the reports of 6 cases of infections of the feet (4 with cellulitis, 2 with gangrene) all of which recovered without operation despite the presence of some degree of circulatory impairment in every case.

It is concluded that the use of protamine zinc insulin in the treatment of diabetes is not contraindicated by the presence of surgical complications.

R. A. M.

ESSENTIAL UNSATURATED FATTY ACIDS IN THE RELIEF OF THE COMMON COLD

By E. F. Boyd and W. F. Connel—*C.M.A.J. Vol.* 37, *No.* 1, *July* 1937, *p.* 38.

Reference is made to some experimental work on rats which indicated the essential nature of certain unsaturated fatty acids in the diet of these animals. From this work there has evolved the concept of a new substance, termed "vitamin F' which is apparently essential for the well-being of animals and man. This substance is associated with and may be identical to an unsaturated fatty acid, linoleic acid.

Information on the clinical value of these essential fatty acids is meagre. However, Oncken reported some work in which it was shown that the serum unsaturated fatty acids tended to fall in periods just preceding the onset of a common cold. He also indicated that feeding oils containing unsaturated fatty acids prevented the onset of colds and elevated the serum unsaturated fatty acids to normal

In the present investigation a systematic study was made to determine if these

unsaturated fatty acids had any real value in the prevention and treatment of the common cold. The preparation used was a purified, concentrated mixture of linoleic and linolenic acids prepared from refined linseed oil. Fifteen minims was arbitrarily chosen as the daily dose after preliminary trials indicated that this amount was well tolerated. A total of 106 young men, mostly medical students, cooperated in this investigation. They were divided into two groups, those who stated they were susceptible to colds (41) and those who were not (65). A period of seven weeks beginning January 1, 1937, was chosen to determine the frequency and duration of colds in each group without treatment with vitamin F. During a second period of seven weeks, each man of group A was given 15 minims daily of vitamin F and no vitamin F was given to the men in group B who were used as controls. The results, which are rather striking are conveniently shown in table form.

Table 1.

Showing the incidence and duration of colds for groups A and B in the control period and test period.

	Average number of colds per man	
	Group A	Group B
Control period	1.54	0.82
Test period	0.56	0.63
	Average duration of each cold	
	Group A	Group B
Control period	11.1	3.8
Test period	2.5	3.8

It would appear that vitamin F resulted in a significant lowering of the incidence and duration of colds in the treated group. Moreover from a study of the individual data it may be calculated that about 27% of the population suffer from a certain number of colds each winter as a result of a deficiency of essential unsaturated fatty acids in the diet.

If this work bears verification by other investigators, it will be a most valuable contribution to medical science.

R.A.M.

A FURTHER REPORT ON THE ASCORBIC ACID TREATMENT OF WHOOPING COUGH

By M. J. Ormerod, B. J. Unkauf, F. D. White —*C.M.A.J. Vol.* 37, *No.* 3, *Sept.* 1937. *p.* 268.

The first report appeared in the August, 1937, number of the same journal and gave an account of 10 cases of whooping cough treated with ascorbic acid. These cases showed that this treatment had an almost specific effect in decreasing the intensity and duration of the disease. In the present communication are presented the results of 17 additional cases of whooping cough treated by oral administration of ascorbic acid. An attempt was made to gain a more accurate idea of dosage and utilization of the vitamin by studying its urinary excretion before and during treatment. By this method a definite hypovitaminosis C was demonstrated in cases of whooping cough and further it was shown that as the body became saturated with the vitamin there was a rapid and marked improvement in the clinical condition of the patient. These clinical changes occurred in the following order:

(1) Marked reduction or complete arrest of vomiting; (2) reduction or disappearance of night cough; (3) reduction in number and intensity of whoop; (4) reduction in number and intensity of day cough.

The change in the general condition of the patient is rapid and clearly noticeable.

The daily dosage adapted was: 350, 250, 250, 200, 200, 150, 150, 125, 125, 100, continuing at the 100 mg. level until the case was complete or stopping the dosage at any stage at which there was a complete remission of symptoms for two days. The average total dose was about 2700 mg. The ascorbic acid is in the form of tablets combined with lactose, ("Redoxon" — Hoffman-La Roche) and will dissolve easily in water, milk or fruit juices or it may be eaten like a candy, as it has a not unpleasant sour taste.

This would appear to be a real advance in the treatment of whooping cough.

R.A.M.

STUDIES WITH HUMAN INFLUENZA VIRUS DURING THE FLU EPIDEMIC 1936-37

By Frances et Al—*J.A.M.A., August* 21, 1937.

This article by Francis et Al, of New York, in view of the uncertainty and controversy as to the etiological agent in human influenza and in view of the widespread interest in virus diseases evident at the present time, is of considerable interest. In this work the virus is transmitted to the nostrils of the ferret in throat washings from the human. Antibody production is studied making use of a mouse protection test and "antibody titre" values obtained by a neutralization test and a complement fixation test. Among the conclusions reached by these workers, may be mentioned the following:

(1) The virus was easily isolated from 80% of cases of human epidemic influenza.
(2) This virus was identical with that obtained in epidemics all over the world since 1933.

(3) There is an increase in antibody titre in convalescence.
(4) These laboratory procedures may be utilized to clarify the variation of clinical picture shown by epidemic influenza.

A.G.

ISOLATION OF TETANUS BACILLI FROM STREET DUST

By Eric C. Gilles, D. P.H. of Baltimore—*J.A.M.A., August* 14, 1937. *p.* 484.

This is an attempt to answer the question, "Is prophylactic treatment with tetanus anti-toxin essential in the case of all persons injured in street accidents?" Samples of street dust were gathered from all parts of the city and 17.4% of these were found to culture tetanus bacilli. The results were taken as proof of the necessity for prophylactic tetanus antitoxin in all cases of street accidents accompanied by abrasion or laceration of the cutaneous surface

A.G.

SYNDROME OF HYPOCHROMIC ANAEMIA, ACHLORHYDRIA AND ATROPHIC GASTRITIS

By Morrison et Al,—*J.A.M.A., July* 10, 1937, *p.* 108.

This is of the nature of a preliminary report but is interesting in that gastroscopic studies are made which give promise of throwing some light on the relationship of gastric conditions to hypochromic anaemia. It may be mentioned that Alvarez and others have suggested that when the haemoglobin falls below about 70% achlorhydria frequently occurs and that with cure of the anaemia, acid may return.

A.G.

CONTROL OF SYPHILIS

By Thomas Parran, *M.D.*, Washington, *D.C.*—*J.A.M.A., July* 17, 1937, *p.* 205.

Dr. Parran facing syphilis as a problem of public health would include it among the official communicable diseases and make it subject to the same laws and regulations as govern them. In addition to the interesting statistics and ideas included in this article, it is of considerable interest to compare Dr. Parran's views with the legislation in force in Ontario in the form of the Venereal Diseases Prevention Act.

A.G.

EDUCATIONAL NUMBER

J.A.M.A., August 28, 1937.

Contains Medical School Survey, Internships and Residencies and information on all aspects of medical education.

CONGENITAL AND PRENATAL SYPHILIS

By H. N. Cole, M.D., Cleveland—*J.A.M.A., Augusi* 21, 1937, *p.* 580.

This is a well written, very interesting and instructive article, dealing with the clinical picture at different ages, the Wasserman reaction, treatment, et cetera and stressing the importance of treating syphilitic mothers throughout pregnancy

A.G.

ARTIFICIAL FEVER THERAPY OF GONORRHOEA IN THE MALE

By Parsons et Al, U.S. Army Medical Corps—*J.A.M.A., July* 3, 1937, *p.* 18.

The conclusions in this article are

(1) Fever therapy accomplishes more cures in gonorrhoea in the male than does chemiotherapy.
(2) Fever therapy greatly reduces the duration of gonorrhoeal infection in the male.

In this article no mention is made of Prontolin which appears to be so much in favour at the present time.

A.G.

THE USE OF EXPECTORANTS

By Charles L. Brown, M.D., Philadelphia *J.A.M.A., July* 24, 1937, *p.* 268.

This article, written on good authority may be of assistance in the choice of "cough medicines" so often required by patients.

A.G.

ACUTE APPENDICITIS IN CHILDREN

By Philip D. Allen, M.D.—*J.A.M.A., July* 10, 1937. *p* 121.

This study of acute appendicitis is based on 612 cases under 13 years of age in the Children's Surgical Service at Bellevue Hospital.

It deals with symptomatology and urgency of operation—operative and non-operative complications. Among conclusions reached may be mentioned:

1. Acute appendicitis infrequent under 5 years.
2. The relatively high death rate in infants due to the frequency of spreading peritonitis.
3. Cathartics hasten perforation.
4. The increase in death rate with delay in operation.

A.G.

TREATMENT OF POLIOMYELITIS

British Medical Journal, July 31, 1937. *p.* 215

An important consideration in poliomyelitis is the treatment of frank paralysis. Accepted treatment during the stage of recovery may be summarized thus: splinting of affected muscles in position of relaxation from the first; physical treatment begun four to ten weeks after acute onset; including graduated exercises; strict recumbency in cases where anti-gravity muscles are involved; continued treatment till recovery is maximal. Yet certain cases, thus treated show incomplete recovery with serious disability. Experience of such a case, truly catastrophic, will lead anyone to welcome a new form of treatment.

Sister E. Kenny of Queensland has evolved a new system for which astonishing results are claimed. Interest and controversy has led to a current investigation by a Government Commission. Her methods are described in a book entitled "Infantile Paralysis and Cerebral Diplegia." She defines the general principles as:

(1) maintenance of a bright mental outlock.
(2) maintenance of impulse.
(3) hydrotherapy and remedial exercises.
(4) maintenance of circulation.
(5) avoidance of the generally accepted methods of immobilization.

The remedial exercises are those usually employed *except that they are begun during the acute phase and given with great frequency.* It is the *abandonment of splinting and recumbency* that is so startling since against all physiological principles.

Yet results count. 600 patients have been treated and a small number of these investigated by a surgeon, Mr. J. V. Guinane who states that all recent cases have been totally and permanently cured and all long-standing cases improved. The data has been robbed of much scientific value through the absence of detailed reports of cases. Yet the medical profession must await eagerly the official report of the Investigating Commission, for no one can be satisfied with conditions as they are at present.

J.A.G.R.

PULP SPACE INFECTION

British Medical Journal, July 24, 1937, *p.* 156.

This is an excellent article, which should be referred to all medical students, on the subject of "Pulp Space Infection." This article is well illustrated with drawings and is too detailed to be summarized. The conclusions are here given and the whole article recommended for reference:

(1) Paper is based on 183 cases of infections of the hand, of which 51 cases were pulp-space infections.
(2) Aching, followed by throbbing and inability to sleep, associated with induration of the pulp, should be established indications for immediate operation in pulp-space infection.
(3) With adequate drainage, inflammation in the pulp readily subsides. If resolution is delayed it is urged that the transfixion operation should be carried out.
(4) Complications in pulp-space infection can be prevented by providing adequate drainage in the early stages.
(5) In a chronic case, it is essential to employ radiological examination before proceeding to treatment.
(6) In the treatment of sequestrum formation and the prevention of taenosynovitis, it is necessary to employ care in operation and, if possible, to conserve the base of the distal phalanx

J.A.G.R.

SPONTANEOUS PNEUMOTHORAX

By J. Gough, M.D., Wales—*Lancet, August* 7, 1937. *p.* 315.

In this article, Gough discusses the views held by various writers on the aetiology of spontaneous pneumothorax.

Little is known of the pathology of benign or simple pneumothorax. Many writers have regarded it as due to rupture of an emphysema bulla near a small area of healed subpleural tuberculosis. Near a scar at the lung apex, there may occur singly or in greater numbers air-containing bullae up to the size of a cherry. These are not to be compared with the bullae of ordinary emphysema.

In addition to the bullae associated with scarring of the lung, there is another type due to congenital malformation. Rupture of these may also give rise to pneumothorax.

Bullae are not present in every case, as in some fatal cases, only adhesions between the collapsed lung and summit of pleura are found.

Non-tuberculous spontaneous pneumothorax may occur in healthy persons, in whom no gross abnormality can be found in the lungs, or may be associated with general emphysema. The latter, however, is rare because the condition usually occurs in healthy young males 20-30 years of age.

There is definite radiological and post-mortem evidence that rupture of a bulla whether due to scarring congenital malformation or to another cause is often responsible for pneumothorax but in the majority of cases these cannot be detected, by any method during life.

Ramsbottom believes that pneumothorax

arises only in cases in which the ruptured bulla is in free communication with a bronchus, large enough and rigid enough to resist collapse.

The part played by exertion in the causation of pneumothorax is difficult to assess. In about one third of cases, it occurs without apparent immediate cause. In approximately another one third, the onset is associated with rather severe bodily exertion. Coughing, sneezing and laughing account for most of the remainder.

Abdominal symptoms are apparently rare, but spontaneous pneumothorax may simulate an acute abdominal condition.

J.D.D.

TUMOURS OF THE URINARY BLADDER

By Terrence Millin—*British Journal of Surgery, July*, 1937.

These are mainly papillomata, papillary carcinomata and infiltrating carcinomata. Using biopsies, Broder classes them as type 1 (¾ of the cells epithelial) type 2 (½ the tumour so differentiated) type 3 (¾ of the cells undifferentiated) type 4 (no differentiation.

The *Symptoms* are those of an intermittent haematuria, in which micturition is painful only when infection is present. Friable papillary growths bleed early. Bladder neck growths cause dysuria; if at ureteric opening, hydronephrosis results. The urine is infected where growth is frankly malignant. In the *Diagnosis* the history is suggestive but cystoscopy is the final arbiter. Experienced men identify induration of bladder base by vaginal or rectal examination. Malignant tumours look solid with an oedematous, necrotic base. For doubtful cases use a diagnostic diathermy; benign tumours will respond. Interior of diverticuli are visualized with flexible lamp. An intravenous urogram will distinguish primaries at ureteric orifice from secondaries in kidney pelvis. In papillary tumours and those about bladder neck examine the prostate and posterior urethra for implants. Cystogram may indicate amount of infiltration but is only used when cystocopy is unsatisfactory. The diagnosis must be made from tuberculous granuloma, tertiary syphilitic ulcer, bullous oedema, etc.

Treatment by *Per-Urethral Coagulation* is best for accessible tumours of low malignancy, and is given once every third week until it is all gone. Patients should be examined regularly after removal. In *Per-Urethral Resection* advocated by the author, after the patient is anaesthetized, adrenalin is injected into the tumour which is then resected with the loop. The base is coagulated and bladder irrigated with 1:5000 silver nitrate. Bulky tumours are first reduced in size with evacuator. This treatment is contraindicated when the tumour is definitely malignant, when it is in a diverticulum, where the growth is extensive, when the tumour is inaccessible or when the prostate is involved. *Suprapubic Resection*: here the bladder is exposed and exteriorized, packed off, and opened with a diathermy needle. Large growths are digitally avulsed. Divide the pedicle of smaller ones with endothermy needle and coagulate the base, then implant radon seeds or apply low voltage X-ray. Fill the viscus with 50% alcohol, suture the bladder about drainage tube. In Partial Cystectomy separate the bladder before opening and if the peritoneum is adherent, cut it and then sew up the defect. With cutting current remove the growth and surrounding half-inch of bladder wall. In Total Cystectomy either subcutaneous Neostomy or bowel implantation of ureters is done first. Bladder is removed three weeks later. For inoperable cases a *Permanent Cystotomy* is done and the haemorrhage is checked by coagulation. *Prognosis* is bad in cases of multiple tumours but they may be well controlled with cystoscopic diathermy. In the open operation for papilloma results have been greatly improved by modern technique but it is too early to assess the ultimate results of per-urethral procedures.

A.G.M.

PATHOLOGY OF THE CERVIX

By Walter Schiller, Vienna.*Amer. Jour. of Obs. and Gyn., Vol.* 34. 1937, *p.* 430.

The earlier a case of carcinoma of the cervix is irradiated or operated upon, the greater is the probability of a permanent cure. With this belief, Schiller, some time ago, devoted his efforts to establish better methods of early diagnosis. Carcinoma grows very slowly at the beginning and offers a good opportunity for diagnosis and eradication.

About 30% of all cases of beginning carcinoma of the cervix give the appearance of leukoplakia and a biopsy in these cases would establish the diagnosis. In the remaining 70%, however, there is no leukoplakia. Normal epithelial cells contain glycogen which stains a deep brown colour when a solution containing iodine is applied. Carcinomatous cells, however, do not stain and thus a latent carcinoma of the cervix may be transformed into a visible one by the application of an aqueous solution of iodine, the carcinoma remaining white while the surrounding epithelium stains a deep brown. This test serves to indicate suspicious spots which should be investigated histologically, removing some of the surface epithelium simply with a sharp curette.

If this test were carried out routinely on all women the probability of healing of all

carcinoma of the cervix could be extended to about ninety-six per cent.

F.C.B.

THE DIAGNOSIS OF THE SEX OF THE HUMAN FOETUS IN UTERO

By Stuart B. Blakely—*Amer. Journ. Obs. and Gyn.*, 1937, *Vol.* 34. *p.* 322.

This article is a review of the various means and methods that have been employed in foetal sex diagnosis and includes the older "superstitions" and some of the more recent investigations.

All efforts made to diagnose the sex of the human foetus may be placed in three groups. In the first group are the beliefs that the male comes from the right side of the uterus or right ovary and that male pregnancies rouse right-sided symptoms in the mother. In the second group are the beliefs that the physical attributes of the foetus during pregnancy and labour differ in the sexes. In the third group are the beliefs that the male foetus, through its secretions affects the mother differently than does the female foetus. Modern investigations of these beliefs have one or more representatives in each group. In the third group, endocrinology and serology have been the means employed, with some encouraging results.

As yet the problem of foetal sex diagnosis has remained unsolved.

F.C.B.

THE EFFECT OF ASCORBIC ACID (VITAMIN C) ON TOXIN PRODUCTION BY C. DIPHTHERIAE IN CULTURE MEDIA

By I. J. Kligler, L. Leibowitz and M. Berman—*The Journal of Pathology and Bacteriology (Great Britain) Vol.* 45. *No.* 2. *September*, 1937. *p.* 415.

It had previously been suggested that the variation in the severity of the results of diphtheria infection from region to region, from species to species and from man to man, was due to variation of the vitamin C content of the tissues of the animal or individual affected. In this paper a series of experiments are reported in which it is shown that the addition of vitamin C to culture media suitable for toxin production renders the filtrates of cultures of this organism grown thereon less toxic (one-twentieth that of controls); diphtheria toxin incubated with ascorbic acid at 30 degrees C loses its toxicity in from two to three days; and diphtheria toxin injected into guinea pigs leads to a decrease in the vitamin C content of the adrenals of the pigs.

W.G.R.

CHANGES IN THE CENTRAL NERVOUS SYSTEM FOLLOWING ARSPHENAMINE MEDICATION

By Dorothy S. Russell—*The Journal of Pathology and Bacteriology (Great Britain) Vol.* 45. *No.* 2. *September*, 1937. *p.* 357.

Three cases of death following arsphenamine treatment are discussed, with a presentation of the gross and histological findings in the central nervous system. In addition to the haemorrhages described by other investigators, non-haemorrhagic, perivascular, demyelinated areas of softening in the white matter are described in two cases. Conjecture as to the cause leads to the suggestion that the phenomenon is one of sensitation, those lesions which are non-haemorrhagic being of a less intense nature. The similarity of the lesions to post-vaccinial encephalitis and the encephalitis seen at times following anti-rabic treatment is also duly noted.

W.G.R.

REACTIVATION OF A PRIMARY TUBERCULOUS COMPLEX AS A SOURCE OF TUBERCULOUS REINFECTION

By Herbert S. Reichle, M.D. and Mae Galavan, M.D.,—*The Archives of Pathology. Vol.* 24, *No.* 2. *August* 1937.

Adult tuberculosis is now thought of as the result of the interaction of tissues previously changed by the effect of a primary tuberculous infection and the tubercle bacillus, whose source is usually exogenous. This paper shows that an endogensus reactivation of the primary tubercle may be the source of the adult type of disease. Seven cases showing evidence of reactivation rather than reinfection are presented and discussed. These were gleaned from eight hundred routine autopsies. The authors believe that endogenous infection should be accepted as a significant mode of extension of pulmonary tuberculosis.

W.G.R.

THE PATHOLOGY AND PATHOGENESIS OF CLINICAL ACUTE NEPHRITIS

By E. T. Bell, M.D.,—*The American Journal of Pathology. Vol.* 13. *No.* 4. *July*, 1937. *p.* 497.

All acute renal diseases with impairment of renal function characterised by retention of nitrogenous products, decreased excretion of phenolsulphonephthalein, inability to concentrate, loss of protein, oliguria or anuria and bleeding from the kidney parenchyma, are included in the series. Benign albuminurias, acute obstructive lesions and acute pyelonephritis are excluded. The cases were

divided into main groups as follows: Acute glomerulo-nephritis — 79 cases; vascular lesions—3 cases; interstitial lesions—2 cases including one of acute lymphatic leukaemia; tubular disease of the kidney—12 cases and extrarenal uraemia—15 cases. The gross and microscopic appearance of these are described and discussed. Recommended.

W.G.R.

STUDIES ON THE MECHANISM OF MORPHINE HYPERGLYCEMIA. THE ROLE OF THE ADRENAL GLANDS

By Bodo, R. C., CoTui, F. W., and Benaglia, A. E.—*J. Pharmacol. & Exper. Therap.*, 61, 48 (1937).

Ten mgm. of morphine sulphate per kgm. dog, or eight mgm. per kgm. cat, given subcutaneously invariably causes a very marked hyperglycemia. The effect is usually noticeable in 15 minutes and reaches a maximum within two hours. An increase in blood sugar from 82 mgms. per 100 c.c. to 167 mgms. in 105 minutes is representative. After denervation and demedullation of one adrenal gland and removal of the other, similar doses of morphine sulphate cause only a very slight hyperglycemia or none at all. These results do not agree with those of Stewart and Rogoff, and those of Houssay, Lewis and Molinelli. However, it is believed that they are more valid than the latter, because of histological control. The conclusion is reached that the adrenal glands play the predominant role in morphine hyperglycemia in both dogs and cats. The slight hyperglycemia which occurs occasionally after adrenal inactivation is attributed to direct action on the liver, to suppression of insulin secretion, or to sympathin production. Later experiments of Bodo and Brooks (same journal, 61, 82, 1937) show that the impulses arousing the sympathico-adrenal system to hyperactivity in normal animals must come from supraspinal centres, since morphine does not directly excite adrenals or lateral sympathetic chains, and actually depresses the spinal cord.

F.L.L.

THE EFFECT OF CHOLINE ON THE LIPID METABOLISM OF BLOOD AND LIVER IN THE COMPLETELY DEPANCREATIZED DOG MAINTAINED WITH INSULIN

By Kaplan, A., and Chaikoff, I. L.—*J. Biol. Chem.*, 120, 647 (1937).

Pancreas contains two factors active in the lipid metabolism of the depancreatized dog maintained with insulin: (1) a heat-labile blood factor, which causes a rise in all blood lipid constituents, especially cholesterol; (2) a heat-stable liver factor, which prevents the deposition of fat in the liver. The daily administration of choline in large amounts or of pancreas prevents the deposition of abnormal amounts of fat in the livers of depancreatized dogs maintained with insulin. However, the curative actions of both choline and pancreas upon livers in which a marked fatty infiltration has already occurred are very slow, many weeks being necessary to demonstrate a measurable decrease in liver lipids. Choline combined in the lecithin of meat does not prevent the deposition of fat and must therefore be less readily available to the animal than is free choline, perhaps due to the absence of pancreatic enzymes. Whether choline is the only active liver factor in pancreas or whether pancreatic action on the liver is due partly to a factor other than choline is left unsettled. It is shown, however, that the factor in pancreas that produces hyperlipemia in the depancreatized dog is not choline, since choline only prevents the occurrence of a hypolipemia.

F.L.L.

REFLEX INHIBITION OF KNEE-JERK FROM INTESTINAL ORGANS

By J. G. Dusser DeBarrenne and A. A. Ward, Jr.—*Amer. Journ. of Physiology, Vol.* 120, *No.* 2.

By means of a mechanical device to insure equal stimulation of the patellar tendon, the investigators were able to demonstrate on anaesthetized monkeys and cats that distension of the urinary bladder, gall bladder or loop of ilium had either partial or total inhibitory effect on the knee-jerk. This effect was considerably more marked in the monkey than in the cat. This inhibition continued after transection of the cord in the mid-thoracic region indicating that it is a spinal reflex.

It was further found that distension of a loop of gut in the monkey was followed by a weak flexion reflex and also that flexion reflexes elicited by electrical stimulation were enhanced by this condition.

It is thus indicated that distension of an abdominal viscus tends to elicit a flexion response which has an inhibitory effect upon the knee-jerk, an extension reflex.

G.D.T.

ROLL OF FAT IN THE DIET.

By Anderson and Williams.—*Physislogical Reviews, July*, 1937.

The first article in the July issue is a particularly comprehensive review of the role of fat in the diet. This subject is dealt with under a number of headings, viz.: historical aspects, general considerations of fat in the diet, fats as carriers of vitamins, essential fatty acids, the vitamin B sparing

action fats, relation of dietry fat to body fat, and the digestibility and absorption of fat. The article concludes with an interesting discussion of the part played by fat in the diabetic diet.

The importance that fat plays in the chemistry and nutrition of the body is a subject familiar to students who have studied biochemistry. The authors of this article have done an excellent piece of work in reviewing the important literature to this date. As such, it is highly recommended to pre-clinical students for special reading.

For those who are interested in the chemistry of the male sex hormones, an article under this heading is to be found in the April issue. An excellent bibliography is to be found following both these articles.

M.E.H.

Book Review

DIET AND HIGH BLOOD PRESSURE.

By Dr. I. Harris, *Honorary Physician, London Heart Hospital.*

This is an interesting little volume inspired by extensive research work by the author and with a specific purpose in view. It is designed to serve as a manual for laymen in acquiring an understanding of the problem and preventive aspects of high blood pressure and heart disease, the prevalence of which is reaching astounding proportions.

The aetiological factors in such conditions are difficult of assay. Insufficient kidney function undoubtedly is a major factor in many cases. The author has found that reductions in Protein intake can greatly decrease the work of the kidneys and so forestall kidney senility. Wisely he points out the folly of such measures during the growing period and advises such restrictions after the age of thirty-five years. Towards an understanding of this, the author has discussed foods and food values, heart and blood-vessel physiology and metabolism. This is done in a clear, simple and accurate manner. Well-planned dietary regimes occupy some fifty pages, making the issue practical indeed.

The less tangible causative factors appear to be bound up in a philosophy of life. Dr. Harris has many inspiring lines to prompt men and women to seek a sound basis upon which happiness and usefulness alone can be substantially built.

A word must be said about the layout of the book. When read as a whole, it does not impress one with its coherence. It is divided into sections, four in all, plus an appendix. Each of these is divided into many subsections of one or two pages, each an essay in itself. This plan undoubtedly makes it a most valuable manual for those who can read for short periods only, or for those whose limited knowledge and training make its assimilation in small portions a necessity.

At $3.50 it represents a sound investment capable of producing huge dividends. The book is published by the Hogarth Press and distributed by Longmans, Green and Co., Toronto, for whom we have been pleased to prepare this review.

H.H., M.D.

RAIN BROS.

For all your

MEDICAL BOOKS
FACULTY STATIONERY
LEATHER NOTE BOOKS
BOND PAPER REFILLS

At Reduced Prices

Free Premiums

RAIN BROS.

353 Yonge Street.

WA. 7385. AD. 6826.

Open evenings. We deliver.

RECENT PUBLICATIONS

Acquired Since June, 1937, by the Medical Reading Room in the University Library

A

Abbott, M.E.: Atlas of congenital cardiac disease. 1936.

Arvedson, J: Medical gymnastics and massage in general practice. et. 4. 1933.

Arvedson, J: The technique, effects and uses of Swedish medical gymnastics and massage. 1936.

Atkinson, F. R. B.: Acromegaly. 1932.

Atlas of human anatomy: with explanatory text by J. F. Williams. 1935.

B

Baborka, C. J.: Treatment by diet, ed. 2. rev. 1935.

Bailey, H. and Matheson, N. M.: Recent advances in genito-urinary surgery. 1936.

Bassett, C.: Mental hygiene in the community. 1936.

Bellingham-Smith, E. and Feiling, A.: Modern medical treatment. 2 vols. 1931.

Berkeley, (Sir) Comyns and Bonney, V. B.: A textbook of gynaecological surgery. ed. 3. 1935.

Boyd, M. F.: Preventive medicine. ed. 5. 1936.

Boyd, W.: An introduction to medical science. 1937.

British Committee on Chronic Rheumatic Diseases: Reports on chronic rheumatic diseases. vols. 1-3. 1936.

British encyclopaedia of medical practice. vols. 1-4. 1937.

Brock, S.: The basis of clinical neurology. 1937.

Brown, E. L.: Physicians and medical care. 1937.

Buck, R. W.: Essentials of physical diagnosis. 1935.

C

Cameron, H. C.: The nervous child at school. 1933.

Christopher, F.: A textbook of surgery. 1936.

Clark, W. I. and Drinker, P.: Industrial medicine. 1935.

Cole, W. H. and Elman, R.: Textbook of general surgery. 1936.

Colyer, (Sir) Frank: Variations and diseases of the teeth of animals. 1936.

Cowgill, G. R.: The vitamin B. requirement of man. 1934.

Craig, C. F.: Amebiasis and amebic dysentery. 1934.

Crew, A. and Gibson, K. W. A.: A dictionary of medico-legal terms. 1937.

D

Davis, L.: Neurological surgery. 1936.

De Kiriline, L.: The quintuplets' first year. 1936.

Dorcus, R. M. and Schaffer, G. W.: Textbook of abnormal psychology.

E

East, W. N.: Medical aspects of crime. 1936.

Eddy, W. H. and Dalldorf, G.: The avitaminoses. 1937.

Elmer, W. P. and Rose, W. D.: Physical diagnosis. ed. 7. 1935.

F

Fearon, W. R.: Nutritional factors in disease. 1936.

Fischer, A. G. T.: Chronic (non-tuberculous) arthritis. 1929.

Fisk, E. L. and Crawford, J. R.: How to make the periodic health examination. 1928.
Fry, H. K.: An introduction to general therapeutics. 1935.

G

Garrod, (Sir) Archibald and others: Diseases of children. ed. 3. 1934.
Grafe, E.: Metabolic diseases and their treatment, translated by Boise. 1933.
Granger, F. B.: Physical therapeutic technic. ed. 2. rev. 1932.
Griffith, J. P. C. and Mitchell, A. G.: The diseases of infants and children. ed. 2. rev. 1937.

H

Hadfield, C. F.: Practical anaesthetics. ed. 2. 1931.
Hall, I. S.: Diseases of the nose, throat and ear. 1937.
Harris, W.: The facial neuralgias. 1936.
Heiser, V. G.: An American Doctor's Odyssey. 1936.
Hendrick, I.: Facts and theories of psychoanalysis.
Herrmann, L. G.: Passive vascular exercises and the conservative management of obliterative arterial diseases of the extremities. 1936.
Hertzler, A. E.: The technic of local anesthesia. 1937.
Hill, C.: A manual of normal histology and organography. 1937.
Hinton, W. A.: Syphilis and its treatment. 1936.
Holmes, E.: The metabolism of living tissues. 1937.
Holmes, G. W. and Ruggles, H. E.: Roentgen interpretation. ed. 5. rev. 1936.
Huddleson, I. F.: Brucella infections in animals and man. 1934.
Hutchison, R. and Mottram, V. H.: Food and the principles of dietetics. ed. 8. 1936.

J

Jameson, W. W. and Parkinson, G. S.: A synopsis of hygiene. ed. 5. 1936.
Jamieson, E. B.: Illustrations of regional anatomy. Sections 1-7. 1937.

K

Kahn, R. L.: Tissue immunity. 1936.
Kerr, J. M. M.: Maternal mortality and morbidity. 1933.
Kerr, J. M. M. and others: Operative obstetrics. 1937.
Krecke, A.: The doctor and his patients. 1934.

L

Levine, S. A.: Clinical heart disease. 1937.
Levy, R. L. (ed.): Diseases of the coronary arteries and cardiac pain.
Luten, D.: The clinical use of digitalis. 1936.

M

McGonigle, G. C. M. and Kirby, J.: Poverty and public health. 1936.
McKenna, R. W.: Diseases of the skin. ed. 3. 1933.
Major, R. H.: Physical diagnosis. 1937.
Manson, (Sir) Patrick: Tropical diseases; ed. by P. H. Manson-Bahr. ed. 10. rev. 1935.
Marriott, W. M.: Infant nutrition. ed. 2. 1935.
Mathews, A. P.: Principles of biochemistry. 1936.
Mekie, E. C.: Handbook of surgery. 1936.
Meredith, F. L.: Twelve hours of hygiene. 1935.
Miller, C. J.: An introduction to gynaecology. ed. 2. 1934.
Mitchner, P. H.: The modern treatment of burns and scalds. 1935.
Muir, (Sir) Robert: Textbook of pathology. ed. 4. 1936.

N

Newburgh, L. H. and Mackinnon, F. H.: The practice of dietetics. 1934.
Nicholson, D.: Laboratory medicine. ed. 2. 1934.

P

Pardo-Castello, V.: Disease of the nails. 1936.
Park, W. H. (ed.): Public health and hygiene. ed. 2. rev. 1928.
Parsons, (Sir) J. H.: Diseases of the eye. ed. 8. 1936.
Practitioners' library of medicine and surgery, vol. 12.—Preventive Medicine. 1937.

R

Rasmussen, A. T.: The principal nervous pathways. 1935.
Ray, M. B.: Rheumatism in general practice. 1934.
Remington, J. P. and Cook, E. F.: Practice of pharmacy. ed. 8. 1936.
Rhinehart, D. A.: Roentgenographic technique. ed. 2. 1936.
Rolleston, (Sir) Humphry: The endocrine organs in health and disease. 1936.

S

Schamberg, J. F. and Kolmer, J. A.: Acute infectious diseases. 1928.
Sollmann, T. H.: A manual of pharmacology. ed. 5. 1936.
Stiles, P. G.: Nutritional physiology. ed. 7. 1931.
Stohr, P. and Lewis, F. T.: A text-book of histology, by Bremer. ed. 5. 1936.

T

Tidy, N. D.: Massage and remedial exercises. 1934.
Topley, W. W. C. and Wilson, T.: Principles of bacteriology and immunity. ed. 2. 1936.
Trease, G. E.: A text-book of pharmacognosy. ed. 2. 1936.

V

Van Dyke, H. B.: The physiology and pharmacology of the pituitary body. 1936.

W

Watson-Williams, P.: Chronic nasal sinusitis. ed. 2. 1933.
Whitney, J. S.: Facts and figures about tuberculosis. 1931.
Whitney, J. S. (comp.): New and supplementary facts and figures about tuberculosis. 1935.
Wolf, H. F.: Textbook of physical therapy. 1933.
Wolf, W.: Endocrinology in modern practice. 1937.
Wolff, E.: The anatomy of the eye and orbit. 1933.
Wolff, E.: A pathology of the eye. 1934.

Compliments of "DOUG LAURIE"

BROWN'S SPORTS & CYCLE CO. LTD.

SPECIAL DISCOUNT TO STUDENTS

345 YONGE ST. at Gould. OPEN EVENINGS WA. 2337